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GOITRE STUDIES: III. THE IODINE PROPHYLAXIS OF ENDEMIC GOITRE.

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THE prevention of endemic goitre in a goitre area entails the intake by the population and particularly by the children of additional iodine. The ideal arrangement would be to supplement each day the quantity present in the food by the amount necessary to bring the total intake up to the requirement needed for optimal health. In practice this is not possible for the quantity present in food and water taken by an individual is unknown, and can be at best only be assessed from general figures.

The prophylactic iodine should be taken fairly frequently in quantities that will ensure a continuously high level of tissue iodine. Recent studies[1] have shown that for three days after a load dose the extra iodine is excreted in the urine, but thereafter the urinary iodine returns to the pre-medication level. This suggests that the prophylactic dose should be given at least at weekly intervals and that it should be large enough to ensure tissue saturation on each occasion.

When it was recognized that Canberra was situated in an endemic goitre area, consideration was given to the most satisfactory method of prophylaxis for the children living in this area.

During the last twenty-five years a number of media have been used to carry iodine. Those advocated by public health authorities in various countries include the following: (i) iodized confectionery; (ii) iodization of water supplies; (iii) iodized milk; (iv) iodized salt; (v) potassium iodide or sodium iodide solutions or tablets.

IODIZED CONFECTIONERY.

At the suggestion of Klinger,[2] and according to the results of tests by Hunziker and von Wyss, chocolate tablets were used as a vehicle for the administration of iodine to school children in Switzerland in 1921. The first tablets contained 10 milligrammes of iodine, but this was reduced in some areas to three milligrammes and even to one milligramme. The iodine was present in the ratio of four parts of iodostarin (an organic iodine preparation) to one part of sodium iodide. Each child received one tablet per week. Wherever the tablets were used, there was a gradual decrease in the incidence of goitre and in the size of existing goitres after one to two years.

In 1925, chocolate tablets containing 0·02 gramme of iodide were introduced into schools in Italy for goitre therapy, again with successful results.

A similar experiment was made in Derbyshire in 1925, iodized sweets being used.[3] Girls whose parents consented were given one sweet per week containing one-tenth of a grain (6·5 milligrammes) of iodine. After six months the incidence of goitre among these girls had increased from 41% to 65%. The reason for the increase is not known. It is worthy of note, however, that 6·5 milligrammes of iodine weekly is a prophylactic dose, whereas a decrease in the incidence of goitre in such a short time could be obtained only by therapeutic means, if at all.

IODIZED WATER SUPPLIES.

Goitre prophylaxis by means of iodized water has been attempted in several endemic areas. The first experiment, and the one which was investigated most thoroughly, was made in Rochester, New York, in the United States of America, in 1923.[4][5][6][7] Sodium iodide, placed in bags, was allowed to dissolve in the water as it flowed from Hemlock Lake into Rush Reservoir, from which the town was supplied. Applications, each of 16·6 pounds of iodide, were made in autumn and spring. At each application iodide was added on twenty-one days, daily for the first week and then on alternate days. The cost was 3000 dollars per year. For the population of Rochester the cost of 3000 dollars brought the annual *per capita* cost to one cent.

By these means the iodine content of tap-water in Rochester was raised to 14 to 28 parts per billion for a period of nearly five weeks twice a year. This corresponds to 14γ to 28γ per litre. Although no apparatus was employed for the dissemination of iodine through the reservoir, it was found that the iodine content of tap-water was uniform throughout the town.

Similar programmes were undertaken in Anaconda[8] and in Saulte Sainte Marie.[9][10] In the latter it was discontinued after a short time as a result of protests from the town's residents. Unfortunately the beneficial effects of these schemes were not measured in terms of the incidence of goitre. The introductory remarks suggest that the dose of iodine in these experiments was too small and was given too infrequently.

The latter objection to the above schemes is not applicable to the experiment made in Derbyshire, England, in 1925.[3] There two pounds of sodium iodide were added to the public water supply each week at a cost of 0·38 pence per person *per annum*. Medical examinations made after six and twelve months showed a decided increase in the incidence of goitre amongst school children, and the programme was discontinued at the end of twelve months. The reason for the failure of the measure was not advanced by the investigator, and as no assays were made of the iodine content of the tap-water it is impossible to make any comment on the scheme.

As a prophylactic measure the iodization of a town's water supply has a number of disadvantages. It is wasteful of iodine, as a large percentage of the water is used for purposes such as sewage and cleaning, for which the added iodine is without benefit. The variation in water consumption presents another difficulty.

It is pertinent to note that the authorities in Rochester, where perhaps the most extensive trials of iodization of a town water supply have been made, considered it only a temporary measure, for the following comment about the scheme appeared in the *Rochester Health Bulletin* of May, 1926:[7]

> . . . that it would be in force until through education or in some other way we get the people to consume iodised salt.

IODIZED MILK.

The use of iodized milk was investigated in Germany as a possible method for the prevention of goitre in young children who do not benefit from the goitre prophylaxis programmes conducted through the schools. It was shown[11] that the iodine content of cow's and goat's milk can be raised by the continuous addition of potassium iodide to fodder. The value of the measure depends on the milk consumption.

IODIZED SALT.

It is obvious that the value of iodized salt as a prophylactic measure against endemic goitre will depend on the degree of iodization of the salt and on the level of salt intake.

History of Iodized Salt.

Europe.

Iodized salt was first used on a large scale in the Canton Appenzell in Switzerland, where salt containing one part of iodine in 100,000 was introduced in 1922.[2] Prior to 1922, 50% of infants in the Canton were born with congenital goitre. After five years it was claimed that there were no cases of congenital goitre among infants whose mothers used the "complete" salt throughout pregnancy.[12] Iodized salt, containing one part of iodine in 200,000, was adopted in the Cantons of Vaud and Nidwalden in 1924, and was later introduced gradually throughout Switzerland.[2] The use of the "complete" salt was not compulsory, but it was recommended by the authorities and sales increased gradually but consistently. Research work in Switzerland in these early years disclosed two important facts: (i) that salt of the concentration of 1 in 100,000 does not eradicate goitre in the most deficient areas, and (ii) that the general use of iodized salt does not increase the incidence of toxic goitre.[2][13]

The example set by Switzerland was followed, in a few years, by other European countries where goitre was prevalent.

New Zealand.

Iodized salt was first used in New Zealand in 1923, when Hercus obtained permission to introduce it at the orphanage of Saint Vincent de Paul in Dunedin.[14] The average daily consumption of salt among these children was found to be two grammes. The salt was iodized to 1 part in 10,000, which means that a theoretical addition of 200γ of iodine per day was provided to each child, without allowance for losses of iodine during cooking. At the time that the iodized salt was introduced, 54 of the 76 children at the orphanage were suffering from thyreoid enlargement. After eighteen months no child who was goitre-free at the beginning of the period had developed a goitre, and of the 36 children who had had goitre, 25 showed a decrease in its size and 11 showed a slight increase in its size. The discrepancy between the numbers of children at the initial and final examinations was not explained.

In 1924 iodized salt was defined in the food and drug regulations for New Zealand as "salt prepared for table or for culinary use by the addition of one part of potassium iodide or of sodium iodide to every two hundred and fifty thousand parts of salt". This salt was to be labelled "iodized". In contrast, "medicated salt", for use under medical direction only, was defined as "salt prepared for table or for culinary use by the addition of potassium iodide or sodium iodide in proportions other than those of iodized salt, provided that in no case shall the proportion be greater than one part of iodide to one hundred thousand parts of salt".[14] In 1932 the definition of "iodized salt" was amended to describe it as salt containing not less than one and not more than two parts of iodide per 250,000. It was stipulated that only packeted salt could be labelled iodized.[15]

By 1936 iodized salt constituted 30% of the salt sold for domestic purposes. Hercus *et alii*[16] confirmed the observation that the urinary excretion of iodine was increased by the regular use of iodized salt. An average consumption of 7.5 grammes of iodized salt per day for cooking and at the table over periods ranging from eight weeks to twelve months raised the iodine excretion of the residents in five institutions in New Zealand by 14% to 85%. In all cases the average urinary iodine excretion of goitrous individuals was raised above the average excretion of non-goitrous individuals prior to the use of the iodized salt.

Despite the successful results of these and other experiments, the annual reports of school medical officers revealed that some districts in New Zealand were still subject to the unaccountable sporadic increases in the incidence of goitre.[15] In 1936 Hercus stated that "the regular use of iodized salt of the present New Zealand standard for domestic purposes is not an efficient protection against goitre".[15]

United States of America.

The first concerted effort for the general use of iodized salt in the United States of America was made in Michigan in 1924 by the Michigan State Medical Society. A survey conducted during 1923 and 1924 had revealed that the incidence of thyreoid enlargement among school children in the Houghton, Wexford, Midland and McComb Counties was 64·4%, 55·6%, 32·7% and 26·0% respectively.[17] By collaboration with manufacturers, a salt containing one part of iodide in 5000 became available throughout Michigan in May, 1924, and after a vigorous campaign the new salt was adopted by about 75% of households.

The scheme received adverse criticism from a group of medical men and the outcry against the widespread administration of iodine was so vehement[18][19] that in 1927-1928 it was considered necessary to investigate thoroughly the effects of the scheme to that date. A repeat survey for thyreoid enlargement was conducted in some of the schools which had been included in the surveys of

1923 and 1924. The incidence of goitre among school children in Wexford and Midland Counties was found to be 17% and 9% respectively, and in the city of Grand Rapids the incidence had fallen from 30% to 9%. No child was found who had suffered ill effects from the use of iodized salt. In addition, clinics were set up in which 1229 adults with long-standing goitres were studied. It was found that 4·1% of persons who had used iodized salt continuously since its introduction were suffering from toxic goitre, while 55·5% of persons who had avoided its use were suffering from toxic goitre.[20] [1]

After more than twenty years' experience the mass of medical opinion in the United States is that salt of the concentration of one part of iodide in 10,000 is most suitable for use in endemic areas.[20] Public health authorities in that country are of the opinion that a fresh campaign is required at intervals of about five years to keep the need for consistent prophylaxis in the mind of the public.[20]

Salt Intake.

Various figures for salt consumption are available, based on sales of salt and production data. By these means the *per capita* salt consumption for the United States and Great Britain has been estimated as from seven to ten grammes per day.[21] More reliable information is available for New Zealand,[15] where total salt consumption, consumption of salt in the home and consumption of table salt have been determined by survey. The results were as follows: (i) the mean *per capita* intake of salt in a number of private households and in one institution was six grammes daily in the household and an additional five grammes daily from manufactured foods; (ii) the mean *per capita* intake of table salt in a residential hostel was 1·5 grammes daily.

The only data on the salt consumption of children were obtained in New Zealand, where it was found that the average daily intake of salt for cooking and at the table by the children in an orphanage was two grammes per head per day.[14]

Survey to Determine Salt Consumption.

The meagre nature of the information on salt consumption was the deciding factor that led to an inquiry into the salt consumption of children and households in Canberra. This survey was made in three parts, as follows: (*a*) A *questionnaire* was sent to the parents of some 1200 children, asking whether the children used table salt and, if so, whether regularly or only occasionally. (Results, Table I.) (*b*) From this group the parents of over 300 children who used salt at the table volunteered to collaborate in an inquiry into the actual salt consumption of these children. Each parent provided a salt cellar which was filled with a known quantity of salt and returned with the instruction that it was for the exclusive use of the child, who must sprinkle the salt on the food and not on the edge of the plate. (Results, Tables II and III.) (*c*) An investigation was made into the quantity of salt used in households for domestic purposes over a period of four weeks. The household consumption was reduced to the daily consumption per adult male, Burnet's conversion factors being used.[24] The results were as follows: number of households, 175; minimum daily consumption, 0·96 gramme; maximum daily consumption, 20·84 grammes; mean daily consumption, 5·47 grammes.

Examination of the data presented in Table I reveals that some children do not use salt at the table. Most of the children who will use salt eventually are using it at the age of five years, and there is a gradual increase until nine years, after which age the percentage of salt users remains fairly constant. About 85% of children over the age of nine years use salt from a salt cellar regularly and 10% of children never use it. The reason for the failure to do so is not evident from the survey, but it was observed that it is not a reflection of the family custom. Many of the older children who did not use table salt had younger brothers and sisters who did use it. It is evident that a campaign for goitre prophylaxis by means of iodized table salt would not benefit 10% of children between the ages of nine and fourteen years, 25% of children aged between five and eight years, and 50% of children aged below five years. Although the incidence of goitre is highest in boys aged between nine and eleven years and in girls aged between twelve and fourteen years, younger children are found with goitre. In the Canberra survey 12·4% of the boys and 22% of the girls aged between six and eight years who were examined had a palpable goitre, and 4·5% of boys and 5·4% of girls had a visible goitre.[25]

Table II shows that the average daily intake of table salt by children who use salt regularly is in the vicinity of 0·8 gramme. The intake of table salt by Canberra children appears to be independent of age. This quantity of salt, if iodized, would provide (*a*) 8·0γ of iodine at a concentration of 1 part in 100,000, (*b*) 80γ of iodine at a concentration of 1 part in 10,000, (*c*) 160γ of iodine at a concentration of 1 part in 5000.

On the basis of the average intake, salt containing one part or more of iodine in 20,000 would be an effective prophylactic measure. However, 20% of the children who use table salt do not use it in sufficient quantities for a concentration of 1 in 10,000 to be safe, and for 8% a concentration of 1 in 5000 might not be effective. This is in addition to the children who do not use table salt at all.

[1] The high figure for the incidence of toxicity among goitre patients conforms with modern theories. Cole[21] expressed the opinion that at least 50% of non-toxic goitres became toxic if left untreated. Other medical men hold a more drastic view. According to Linnell,[22] by middle life goitres without some evidence of toxicity are rare.

TABLE I.

Numbers and Percentages of Children Who Use Salt at the Table.

Age in Years.	Number of Children.	Salt Used.		Salt Not Used.		Salt Used Occasionally.	
		Number.	Percentage.	Number.	Percentage.	Number.	Percentage.
1	36	6	16·7	27	75·0	3	8·3
2	39	19	48·7	16	41·0	4	10·3
3	60	31	51·7	21	35·0	8	13·3
4	86	48	55·8	28	32·6	10	11·6
5	127	96	75·6	20	15·7	11	8·7
6	129	86	66·7	31	24·0	12	9·3
7	116	90	77·6	18	15·5	8	6·9
8	108	85	78·7	11	10·2	12	11·1
9	95	81	85·3	10	10·5	4	4·2
10	96	80	83·3	13	13·6	3	3·1
11	76	66	86·8	6	7·9	4	5·3
12	92	75	81·5	10	10·9	7	7·6
13	64	60	93·7	3	4·7	1	1·6
14+	117	94	80·3	16	13·7	7	6·0
	1241	917		230		94	

The Stability of Iodized Salt.

If iodized salt were used for all purposes, the percentages quoted in the preceding section would be reduced considerably. However, salt used in the cooking and preparation of food cannot be regarded as a reliable medium for carrying iodine. Although no exhaustive study has been made of the stability of iodized salt used in cooking, there is evidence that some at least of the iodine in vegetables is leached into the cooking water.[29]

During recent years several investigations have revealed that the iodine content of iodized salt is often considerably below the stated amount.[26][27] This has been found to be due to a number of factors. Manufacturers have great difficulty in mixing the iodide uniformly throughout the salt. Iodine is lost on storage, especially in a damp atmosphere, and iodide decomposes unless stabilizers have been added to the salt.

TABLE II.

Daily Salt Intake of Group of Canberra Children Who Used Salt at the Table

Age.	Number of Children.	Minimum. (Grammes.)	Maximum. (Grammes.)	Mean. (Grammes.)
Boys:				
5 years ..	13	0·100	1·59	0·778
6 years ..	18	0·210	1·88	0·798
7 years ..	12	0·370	1·27	0·797
8 years ..	13	0·340	1·50	0·619
9 years ..	19	0·130	2·27	0·854
10 years ..	17	0·070	2·00	0·937
11 years ..	17	0·062	1·30	0·654
12 years ..	9	0·350	2·16	0·978
Girls:				
5 years ..	16	0·260	1·40	0·606
6 years ..	28	0·140	4·33	1·033
7 years ..	14	0·250	1·16	0·690
8 years ..	28	0·030	1·16	0·701
9 years ..	28	0·030	2·40	0·952
10 years ..	22	0·100	1·91	0·845
11 years ..	11	0·070	1·38	0·752
12 years ..	14	0·090	1·33	0·703

THE USE OF POTASSIUM IODIDE OR SODIUM IODIDE IN SOLUTION OR TABLETS.

The use of potassium iodide or sodium iodide in solution or in tablets is essentially the same as the iodized confectionery method, in that measured quantities of iodine are administered at regular times.

The method was used first by Marine and Kimball in the classical experiment with school children in Akron, United States of America.[28] In this, each child who received the treatment was given two grammes of sodium iodide in solution each autumn and spring. The dose was distributed over a period of two weeks. After two and a half years only five out of 2190 children had developed an enlarged thyreoid gland, while 495 children in a control group of 2305 who had received no treatment had developed a goitre. A striking feature of the scheme is the high mean daily dose of iodine.

A modification used in New Zealand was the weekly distribution to school children of one grain (0·065 gramme) potassium iodide tablets[14] The maximum beneficial effects of this programme were not observed for some considerable time, even up to three years after its introduction.

GOITRE PROPHYLAXIS IN CANBERRA.

The foregoing review of the experiences in other countries and the results of the investigation into salt consumption by children in Canberra brought out the following facts.

1. The iodization of a town water supply wastes considerable quantities of iodine, and although it is comparatively cheap, the results of experiments overseas were conflicting. A further disadvantage for the Australian Capital Territory is that a number of villages and most of the farms are not served by the reticulated water supply. For the scheme to be effective, the iodine content of the tap-water must be maintained at a level that will ensure that young children, whose consumption of water is low, will receive somewhere about 100γ a day. This would require a lengthy experiment with measurements of the urinary excretion of iodine over twenty-four hour periods by children of all ages.

2. Iodized milk has not been tried extensively anywhere, largely because its success is dependent upon the milk consumption of each child. In general principles it is relatively more expensive to pass nutrients through animals to humans than to give them directly to the humans.

3. To recommend the use of iodized salt as a prophylactic measure for endemic goitre would give to parents a false sense of security. The facts that many young children do not use table salt and that others use it sparingly mean that iodized salt restricted to table use only would not protect these children. The stability of iodine in cooking salt has not been studied sufficiently to give confidence to this as a reliable source of iodine. To depend upon iodized table salt as the means of goitre prevention places the full responsibility upon the individual parent to have sufficient interest in the welfare of her children to buy and use iodized salt regularly.

TABLE III.

Frequency Distribution of Table Salt Consumption.

Class Interval 0·20 Gramme.	Boys.	Girls.
0·01 to 0·20	8	14
0·21 to 0·40	13	22
0·41 to 0·60	18	30
0·61 to 0·80	30	20
0·81 to 1·00	19	29
1·01 to 1·20	10	18
1·21 to 1·40	5	12
1·41 to 1·60	8	7
1·61 to 1·80	3	1
1·81 to 2·00	2	4
2·01 to 2·20	1	1
2·21 to 2·40	1	2
2·41+	—	1
Total..	118	161

4. The success of Marine and Kimball's original experiment and that of similar trials in Europe and New Zealand indicated that a dose of iodine to the individual child at sufficiently frequent intervals was the most satisfactory measure to adopt for Canberra and its environs. It was realized that the success of the procedure depended upon the full cooperation of the organizations used to distribute the iodine; of this we felt assured.

The Method Adopted for Canberra.

The choice of a vehicle for the individual dose of iodine rested between confectionery and a medicinal tablet. For the obvious reason the latter was selected. The dose to be given was decided after a series of preliminary tests. Following Marine and Kimball's lead a number of children were given two grammes of potassium iodide in solution spread over fourteen days. At least 25% of parents reported some probable toxic effects of this dose. Other children were given 10 milligrammes of potassium iodide once a week without showing any signs of iodism.

The weekly dose of 10 milligrammes is well above seven times the probable daily requirement of iodine of 70γ, but it allows for the rapid excretion in the three days following the dose. Studies are in progress of the daily urinary excretion of iodine of children taking this dose, to ascertain whether at the end of the week the tissues are reasonably saturated with iodine. The results will be reported in a subsequent paper in this series.

As a result of these investigations it was decided to recommend to the Commonwealth Government that it should sponsor a programme for the free distribution of tablets containing 10 milligrammes of potassium iodide to all children between the ages of one and fourteen years resident in the Australian Capital Territory (Canberra and

its environs). In September, 1947, the Minister for Health, the Honourable N. E. McKenna, announced that the programme had been adopted. It came into force in October. Tablets containing 10 milligrammes of potassium iodide have been made available, free, to the children and to pregnant and lactating women living in the Australian Capital Territory.

By courtesy of the New South Wales Director of Education (who is responsible for the administration of the technical aspects of education in the Australian Capital Territory), tablets are administered to school children under the supervision of teachers at school. Tablets for infants, for pre-school children, and for lactating and pregnant women are at present obtainable from mothercraft centres.

Although parental consent is necessary before school children are given tablets, it is gratifying to report that the tablets are now being used by at least 95% of children in Canberra.

SUMMARY.

1. An outline of the results which have been achieved in Europe, New Zealand and America by five methods of goitre prophylaxis has been given.

2. The results of a survey of salt consumption in Canberra are reported. It was found that about 10% of school children over the age of nine years did not use table salt. The average daily intake of table salt by children who use it was found to be 0·8 gramme. The mean daily consumption of common salt by households was found to be 5·5 grammes per adult male.

3. The advantages and disadvantages of the five methods of prophylaxis are discussed.

4. The goitre prophylaxis campaign by the use of ten-milligramme tablets of potassium iodide, which was introduced in the Australian Capital Territory in October, 1947, is reported.

REFERENCES.

(1) N. Nelson, E. D. Palmes, C. R. Park *et alii*: "The Absorption, Excretion and Physiological Effect of Iodine in Normal Human Subjects", *The Journal of Clinical Investigation*, Volume XXVI, 1947, page 301.

(2) W. Silberschmidt: "The Prophylaxis of Endemic Goitre", Report of the International Conference on Goiter in Berne, 1927, page 471.

(3) W. N. Ash: "Goitre", *Journal of State Medicine*, Volume XXXIV, 1926, page 627.

(4) R. Olesen: "Iodization of Public Water Supplies for Prevention of Endemic Goiter", *Public Health Reports*, Volume XLII, 1927, page 1355.

(5) J. W. Elmms: "Iodine Treatment of Water to Prevent Goiter", *Water Works*, Volume LXI, 1924, page 382.

(6) B. C. Little: "Sodium Iodide Treatment of Rochester's Water Supply", *Journal of the American Water Works Association*, Volume XII, 1924, page 68.

(7) A. B. Willis and G. W. Goler: "Iodide in Rochester's Water Supply", *Bulletin of the Health Bureau*, May, 1926, page 3.

(8) "Montana Fights Goiter with Sodium Iodide", *Engineering News Record*, Volume XCVII, 1926, page 378.

(9) H. A. Sherman: "Iodine Treatment of Sault Ste. Marie (Mich.) Water", *City Manager Magazine*, December, 1923.

(10) E. D. Rich: "Iodine Treatment in Michigan", *Journal of the American Water Works Association*, Volume XIV, 1925, page 325.

(11) E. Maurer: "Goiter Prophylaxis with Iodized Milk in Infancy", *Zeitschrift für Kinderheilkunde*, Volume XLIV, 1927, page 120; abstracted in *The Journal of the American Medical Association*, Volume LXXXIX, 1927, page 2077.

(12) G. Muggia: "The Prophylaxis of Endemic Goiter", Report of the International Conference on Goiter in Berne, 1927, page 459.

(13) W. Von Jauregg: "Prophylaxis of Endemic Struma", Report of the International Conference on Goiter in Berne, 1927, page 444.

(14) C. E. Hercus, W. N. Benson and C. L. Carter: "Endemic Goitre in New Zealand and its Relation to the Soil Iodine", *Journal of Hygiene*, Volume XXIV, 1925, page 321.

(15) C. E. Hercus and H. D. Purves: "Studies on Endemic and Experimental Goitre", *Journal of Hygiene*, Volume XXXVI, 1936, page 182.

(16) C. E. Hercus, H. A. A. Aitken, H. M. S. Thomson and G. A. Cox: "Further Observations on the Occurrence of Iodine in Relation to Endemic Goitre in New Zealand and on Iodine Metabolism", *Journal of Hygiene*, Volume XXXI, 1931, page 493.

(17) R. M. Olin: "Iodine Deficiency and Prevalence of Simple Goiter in Michigan", *The Journal of the American Medical Association*, Volume LXXXII, 1924, page 1328.

(18) C. L. Harstock: "Iodised Salt in the Prevention of Goiter", *The Journal of the American Medical Association*, Volume LXXXVI, 1926, page 1334.

(19) O. P. Kimball: "Induced Hyperthyrodism", *The Journal of the American Medical Association*, Volume LXXXV, 1925, page 1709.

(20) O. P. Kimball: "Iodized Salt for the Prophylaxis of Endemic Goiter", *The Journal of the American Medical Association*, Volume CXXX, 1946, page 80.

(21) W. H. Cole, D. P. Slaughter and L. J. Rossiter: "Potential Dangers of Nontoxic Nodular Goiter", *The Journal of the American Medical Association*, Volume CXXVII, 1945, page 883.

(22) J. W. Linnell, G. Keynes and J. E. Piercy: "Some Vulgar Errors in Regard to Goitre", *British Medical Journal*, Volume II, 1946, page 449.

(23) Memorandum of the Goitre Sub-Committee, Medical Research Council of the Privy Council, *The Lancet*, Volume CCXLVI, 1944, page 107.

(24) E. T. Burnet and W. R. Aykroyd: "Nutrition and Public Health", *Quarterly Bulletin of the Health Organisation of the League of Nations*, Volume IV, 1935, page 323.

(25) F. W. Clements: "Goitre Studies: I. The Incidence of Endemic Goitre in Three Areas in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1948, page 637.

(26) C. E. Hercus and K. C. Roberts: "The Iodine Content of Foods, Manures and Animal Products in Relation to the Prophylaxis of Endemic Goitre in New Zealand", *Journal of Hygiene*, Volume XXVI, 1927, page 49.

(27) F. C. Bing: "Some Problems Pertaining to Iodized Salt", National Research Council, Food and Nutrition Board Bulletin, Volume I, 1941, page 162.

(28) D. Marine and O. P. Kimball: "The Prevention of Simple Goiter in Man", *The Journal of the American Medical Association*, Volume LXXVII, 1921, page 1068.

THE DIFFICULT AUTOPSY.[1]

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IT is disconcerting to the pathologist to perform a post-mortem examination and still be unable to determine the cause of death. Such a situation has confronted me from time to time in the course of several thousand examinations, and I have had perforce to develop a technique to meet it.

How often this occurs depends on how easily one's inquisitiveness is satisfied. Post-mortem diagnosis has a fundamental limitation which in some cases renders complete explanation of the death unattainable and some compromise with practicality essential. The act of dying is a process, not a state; it belongs to pathological physiology rather than to morbid anatomy. Yet at an autopsy it is from the morbid anatomy that we attempt to deduce the cause of death. An anatomical finding is significant only in so far as it can be interpreted as evidence of a fatal process. And the anatomical evidence will often be insufficient to warrant an assured conclusion.

The point may be exemplified by atheroma of the coronary artery, a common cause of sudden death. In some cases the atheroma is accompanied by a thrombus completely occluding the lumen or by obvious signs of acute infarction in the myocardium; in these cases the coronary disease is clearly the cause of death. But one often sees considerable narrowing of the lumen of a coronary artery without thrombosis or infarction. If, after a thorough study of the cadaver, this is the only significant post-mortem finding, one is justified in accepting the coronary disease as the presumptive cause of death and in signing the death certificate accordingly. For an undisclosed thrombus in a branch of the artery too small to be dissected may have initiated an abnormal rhythm; the lumen may have been too narrow to supply the necessary blood at a time of exertion or emotion; or spasm may have completed the occlusion.

Yet these coronary arteries had been just as narrow, or almost so, for months. Their narrowness does not explain why death occurred at that moment. One sees from time to time equally narrow coronary arteries in subjects killed by accident, and it may be that my list of cases in which death was ascribed to coronary disease

[1] Read at a meeting of the Mater Hospitals Clinical Society on September 20, 1946, at Brisbane.

includes a few in which the real cause of death was not recognized.

Similarly, when one finds in a case of sudden death considerable hypertrophy of the left ventricle and of arterioles generally, without other disease, it is reasonable to presume that death occurred because the heart, already handicapped by hypertension, was unable to meet an extra demand; but in some of these cases also the presumption may be erroneous.

Even when the immediate cause of death is clear, the steps leading up to it may be quite obscure. Indeed, there is unlimited scope for seeking to clarify the sequence of events from the first morbidity to the fatal conclusion.

One should explain that these examinations were not routine hospital autopsies in which the results of clinical investigations were available to guide the pathologist. They were carried out by direction of the coroner, and therefore included many cases in which a person apparently in good health died suddenly or was found dead; there may have been only a meagre history of illness or none at all. Other cases were referred to the coroner by the clinician because of the obscurity of the symptoms; or there was a history of accident or suspicion of foul play.

STEP 1. COMPLETE THE ANATOMICAL DISSECTION.

Every pathologist has his own routine technique whereby he examines the various structures of the body in a regular, systematic order. This technique will be more or less complete according to the circumstances in which the examination is made, and its purpose. The purpose of a coroner's autopsy is to determine the cause of death, and the government medical officer, who interrupts a busy clinical practice to perform one, will be content to stop when the cause of death has been demonstrated. On the other hand, at a pathological institute each autopsy will be regarded as a research study, every organ thoroughly scrutinized and examined histologically as a matter of course, and the full resources of the biochemist and bacteriologist utilized. It is not to the research pathologist that these notes are addressed, but to the occasional pathologist, the junior pathologist and the harrassed government medical officer.

A common autopsy procedure is to open the thorax and abdomen by a mid-line incision from jugular notch to pubes and remove and dissect the contained organs. If the cause of death is not apparent, the cranial cavity is opened and the brain examined. If the cause of death is still unexplained, we reach the stage at which the occasional pathologist finds himself in difficulties. When this happens the first procedure is to make sure that the dissection is anatomically complete, that all significant organs have been examined. This sounds almost too obvious to mention; yet in practice, with the telephone clamouring for one's presence elsewhere, I know how easy it is to slip into short cuts.

The Throat and Neck.

The throat and the structures of the neck occasionally hold the secret of the death, and if they have not already been included in the routine dissection, they should now be examined. This is conveniently done by making an incision down each side of the neck from the mastoid process along the sternomastoid muscle to the *manubrium sterni*. The skin covering the front of the neck is then reflected up, and the floor of the mouth with the tongue, the soft palate, pharynx, larynx, trachea and œsophagus, together with the anterior muscles of the neck (also the lungs if they have not already been removed), is excised in one piece. These structures are then examined in turn.

There are four groups of cases in which examination of the organs of the neck is most important and should never be omitted: (i) when respiratory obstruction may have occurred, as in drowning, suffocation, choking and death during anæsthesia; (ii) when poisoning is suspected, so that any irritation or corrosion of the pharynx or œsophagus may be observed; (iii) in the examination of young children, in whom inflammation of the throat is common and obstruction of respiration by food or foreign bodies may occur; (iv) when there may be injury to the cervical segment of the spine (as for instance in the case of cyclists thrown forward onto the road), or to the soft tissues of the neck, as in throttling. In these cases the neck organs should not, of course, be examined as an afterthought, but as part of the prearranged post-mortem plan.

In the following cases, examination of the structures of the neck proved all-important:

Obstruction of Airway by Food: Post-Mortem Examination 1372.—A man, aged fifty-eight years, was having his evening meal at a café. He was noticed to collapse and slump back in his seat. The ambulance was sent for, but before it arrived he was dead. Autopsy showed that death was due to suffocation. There was nearly half a pork sausage in one piece in the pharynx, and smaller pieces of sausage were present in the larynx. It was learned that he had been drinking rum at intervals throughout the day. The alcohol had made two contributions to his death; it had abolished his table manners so that he gulped his food, and it had narcotized his cough reflex. He had died without any sign to indicate to those near by that he was choking.

Similar cases of choking by food may be observed in epileptics and in children.

Laryngeal Diphtheria: Post-Mortem Examination 2528.—An infant, aged twenty-two months, died unexpectedly. He was teething and had had a cold for several days followed by a severe cough. Diphtheritic membrane was found *post mortem* covering the tonsils and adenoid tissue, lining the larynx and extending down the trachea to within one inch of its bifurcation. Death was due to respiratory obstruction and bronchopneumonia.

Fracture of Odontoid Process: Post-Mortem Examination 244.—A seaman, aged fifty-four years, was working in a bos'n's chair painting the mast of his ship when the rope apparently became unhitched and the chair fell twenty feet onto a winch. The seaman was dead when his mates reached him. Externally he was found to have a laceration of the right side of the forehead and abrasions on the left leg and sacrum. Although force had obviously been applied to the head, no fracture of the skull was found, nor any macroscopic evidence of injury to the brain. Examination of the cervical portion of the spine revealed a fracture of the odontoid process of the axis, the consequent injury to the spinal cord accounting for the immediate death.

Other Neck Conditions.—Other conditions in the neck which may cause death are retropharyngeal abscess, œdema of the glottis and compression of the trachea by an enlarged thyreoid.

Pulmonary Embolism and Thrombosis.

Pulmonary embolism and thrombosis are an occasional cause of sudden or unexpected death. Massive embolism is best detected by slitting up the main pulmonary artery as far as its bifurcation while the heart is still *in situ*. Less extensive embolism or thrombosis will become evident when the branches of the pulmonary arteries are opened during the dissection of the lungs.

Other Investigations.

The state of the bone marrow should be investigated. The red marrow is most conveniently exposed by a tangential saw cut through the bodies of the lower lumbar vertebræ. It should also be studied in ribs and sternum. The removal and longitudinal bisection of a femur will show, by the relative amounts of red and fatty marrow, if there is abnormal hæmatopoietic activity. In infants a femur should be examined as a routine measure. This may disclose congenital syphilis, rickets or other disorder of ossification.

Calculi in the common bile duct may be overlooked, although their presence will usually be manifest by their effects on liver or gall-bladder. Before the liver is removed the second part of the duodenum should be opened and the orifice of the common duct observed while the gall-bladder is compressed. Failure of the bile to gush forth will indicate some obstruction in the bile passages, which should be investigated further.

Similarly calculi should be sought in the ureters, for sudden death has been ascribed to shock so caused.[1] I have not recognized such a case.

Removal of the spinal cord for examination is laborious and is rarely carried out in the investigation of sudden or unexpected death. I have no example to offer in which such an examination proved significant. Yet death could occur rapidly from hæmatomyelia or fulminant poliomyelitis affecting the cervical region. Appropriate investigation of the cord will be made when indicated by a history of symptoms referable to it or by the finding of trauma to the spine.

The suprarenals merit careful macroscopic examination (followed up by microscopic examination if necessary), as fatal lesions—hæmorrhage, tuberculosis, cortical atrophy—may be quite small.

Other sites which may yield significant information are the middle ears, the dural sinuses (especially in infants), the hæmorrhoidal areas, and the large veins of the pelvis and lower limbs.

STEP 2. CHEMICAL INVESTIGATIONS.

When the complete macroscopic examination of the cadaver has failed to reveal the cause of death, specimens should be taken for chemical, histological and bacteriological investigations.

Chemical examination of the urine may solve the problem of the cause of death. Simple tests for sugar and acetone bodies should be made when necessary at the time of the autopsy, and the necessary facilities should be available in the mortuary.

Death from diabetic coma is not associated with macroscopic pathognomonic signs in the organs, and in cases in which no history is forthcoming this condition is likely to be overlooked if the urine is not examined. In other cases subjects of diabetes die from coronary disease, and I suspect that in many such cases I have not recognized the diabetic factor through failure to examine the urine *post mortem*.

In one case which had eluded clinical diagnosis the only clue found at autopsy was the abnormally dark colour of the urine found in the bladder. Chemical investigation showed this to be due to porphyrins and established the diagnosis of acute porphyria.[2]

Another simple test, which should when indicated be carried out in the post-mortem room, is the Prussian blue test. When a blood dyscrasia is suspected, a thin slice of liver is washed to remove blood, then immersed in a 2% solution of potassium ferrocyanide for a few minutes, then in hydrochloric acid (2%). A blue colour indicates excessive deposition of iron, of which pernicious anæmia and repeated blood transfusions are common causes.

Examination for Poisons.

Certain viscera should be placed in clean glass jars for submission to the analyst, as only by chemical analysis can the presence of some poisons be determined. Morphine, strychnine, atropine and cocaine, for instance, give no characteristic post-mortem signs, though there may be some non-specific features such as œdema, congestion or petechiæ. Arsenic may cause what appears to be infective gastro-enteritis. Other poisons may give hints of their presence, but need the analyst for precise diagnosis. (The two poisons most commonly used suicidally in Brisbane do not as a rule require analysis for their detection; carbon monoxide is evident from the cherry-red colour of the blood and "Lysol" from the smell of the stomach contents.)

Mr. L. A. Meston, F.A.C.I., the late Queensland Government Analyst, to whom and to whose staff I am indebted for the solution of many mysterious deaths, recommends that the following specimens be submitted when examination for poisons is desired: (*a*) the entire stomach with contents; (*b*) the upper half of the small intestine (including the duodenum) with contents; (*c*) the kidneys, the spleen and about half the liver; (*d*) all urine found in the bladder; (*e*) any vomitus; (*f*) the first stomach washings (if the stomach was washed out before death); (*g*) other organs, if indicated by the circumstances.

The police reported the following history.

Strychnine Poisoning: Post-Mortem Examination 437.—A woman, aged thirty-seven years, had always been of a nervous temperament and had had a miscarriage induced eighteen months previously because of acute mania. On the day of her death she seemed in normal health. At 5.30 p.m. her husband came home and had tea with her; he then returned to work at 6.40. At 6.45 she went to the lavatory, and on leaving it a few minutes later said that she was feeling ill and that her legs were numb. At 7.20 she became worse, gasped for breath, commenced to have "fits of violent trembling" (without pain) and died at 7.30 p.m.

Autopsy showed that she was four months pregnant. There was a great deal of congestion and œdema of the lungs, and petechiæ were found in the pleura and heart. As there were no signs to indicate the cause of death, specimens were submitted to the analyst, who reported that strychnine was present in quantity in the viscera and in the stomach contents. The nature of the "fits of violent trembling" was now manifest, but how or why the strychnine came to be ingested was never answered. No container for it could be found in a search of the house and surroundings, nor any record of its purchase.

Alcohol in Blood and Urine.

The analyst may further help the puzzled pathologist in appropriate cases by estimating the amount of alcohol in the blood and urine. A strong smell of some liquor in the stomach indicates that the subject has been drinking, but does not prove that he has taken a fatal quantity. Some exact information from the analyst will, therefore, be welcome.

With regard to the interpretation of the alcohol figures, McNally[3] offers some practical guidance:

> If there is no serious pathology and the blood contains more than 0·43% alcohol . . . one may fairly assume that death resulted from acute alcoholism. If, on the other hand, one finds serious pathology such as extensive cardio-circulatory disease or liver or kidney lesions, it would be fair to assume that in the presence of lesser concentrations of alcohol (0·25% to 0·35%) death was due to a combination of the pathologic lesions found and acute alcoholism.

McNally's figures for alcohol in blood were calculated on a weight/volume basis.

Death from uncomplicated acute alcoholic poisoning is uncommon, for at the usual rate of drinking, coma is likely to supervene before a fatal dose is ingested. It can occur when a large amount of spirits is consumed rapidly, as for a bet, or in drinking competitions. Several examples have come under my notice from the drinking of methylated spirits.

Acute Alcoholic Poisoning: Post-Mortem Examination 1662.—One Sunday morning an elderly woman purchased and brought home a pint of methylated spirits. Before she could drink it, it was taken from her. She went out and obtained another, and this time made sure of it by drinking it on her way home. Soon after reaching home she became unconscious, and six hours later she died in coma. The blood taken at autopsy contained 0·61% alcohol (weight/volume).

Further Observations on Alcohol.

More commonly acute alcoholism is combined with chronic alcoholism. In a subject whose organs have been damaged by years of drinking, I have accepted, following McNally's figures, the finding of more than 0·25% of alcohol in the blood as evidence that acute alcoholism may have contributed to the death. Methylated spirits reveals its presence by its characteristic odour. Several times, however, when this has been evident in the organs at autopsy, subsequent analysis has proved the absence of alcohol from the blood. Of course it is not alcohol, either ethyl or methyl, which gives methylated spirits its odour, but pyridine, and this persists in the body much longer than alcohol. The level of alcohol in the urine is not so significant as that in the blood; it bears no constant relation to the latter, being either greater or less; it indicates, not the alcoholic state of the subject at the time of death, but the average state during the period (usually unknown) in which the urine was collecting in the bladder. Nevertheless, it is often desirable to estimate

the urinary alcohol content as a useful confirmation of the more significant blood alcohol figure.

Blood Chemistry.

Estimation of the urea in blood is of value occasionally in clarifying the cause of death from uræmia.

Estimation of glucose in post-mortem blood has not been attempted in this series. Dr. J. I. Tonge informs me that such an estimation is attended by several fallacies, particularly by the appearance in the blood after death of other reducing substances and by the diffusion of glucose from the liver into the blood in the right side of the heart. Hamilton-Paterson and Johnson[4] found that hyperglycæmia may be diagnosed if a blood sugar value of 200 milligrammes per 100 millilitres or more is found after death in the left side of the heart, and that hypoglycæmic coma as a cause of death cannot be confirmed unless the blood is examined within two hours of death. Further investigations appear to be required before reliance can be placed on post-mortem blood-sugar estimations.

STEP 3. HISTOLOGICAL INVESTIGATIONS.

The next step is to take specimens for histological examination. Here, again, there must be a compromise between research urges and practicality. Few pathologists in this country outside university departments could emulate my American army friend—a first-class morbid anatomist—who as a routine procedure took 35 pieces of tissue at each autopsy. Where facilities for preparation of sections are more limited, a wise selection of tissues is desirable. I regard the minimum for a "problem" post-mortem examination as seven—cerebral cortex, *medulla oblongata*, heart, right and left lungs, liver and kidney. To these should be added any other tissues if suspicion of their morbidity arises. A section of bone marrow will be included if there is a possibility of some disorder of hæmatopoiesis. In addition to the paraffin sections of heart, liver and kidney, frozen sections of these tissues should be prepared and stained for fat.

Microscopic examination of cerebral tissues may reveal syphilis, encephalitis, cerebral malaria, degenerations (as in chronic alcoholism) or vascular disease with its sequels. A tumour of the infiltrating kind, inconspicuous macroscopically, may be found, or a tumour as the underlying cause of a hæmorrhage.

The medulla is highly significant in its relation to sudden death, as it contains the cardiac and respiratory centres, in which a lesion of microscopic size may be rapidly fatal.

The diagnosis of syphilis of the nervous system usually requires the microscope for its confirmation, and may readily be missed if this is not used. When it has caused death, however, there is as a rule some macroscopic hint of its presence—thrombosis of an artery, thickening of the pia-arachnoid, general atrophy of the gyri or granular ependymitis—which will lead the pathologist to take blocks for section as well as blood or cerebro-spinal fluid for serological examination.

Acute Encephalitis: Post-Mortem Examination 3101.—A girl, aged ten years, who had been normal during the previous day, had a bout of coughing during the night. At 4.30 a.m. she had an attack of "croup". In the ambulance she had a "hæmorrhage" (probably a flow of blood-stained, œdematous fluid) from nose and mouth, and death had occurred before her arrival at the hospital at 5.30 a.m.

The lungs at autopsy were congested and œdematous, and fine froth filled the bronchi. The mucosa of the larynx was congested. There were a few petechiæ in the epicardium and some congestion of the brain. *Corynebacterium diphtheriæ* did not grow in attempted cultures from nose and throat. Histological examination revealed cellular foci in the medulla (probably consisting of glial cells and lymphocytes) and perivascular demyelinization in the white matter of the cerebrum.

The Heart.

Histological study of the heart often illuminates the cause of sudden death. One may find unsuspected inflammation or acute infarction, narrowed arteries, or fine fibrosis indicating slow starvation of the myocardium. A frozen section, suitably stained, may reveal fatty degeneration of muscle fibres. Another occasional finding is excessive fatty infiltration of the wall of the right ventricle, which could perhaps lead to failure of the right side of the heart.

Myocarditis, Probably Diphtheritic: Laboratory Number 86360.—A female baby, aged ten and a half months, who had been well, became listless one night and increasingly so next day, and died suddenly in the afternoon. The passage of one offensive stool caused suspicion of gastro-enteritis. No abnormality was found at the post-mortem examination except congestion of the lungs, liver, kidneys and suprarenals. The tonsils were small, and there was no sign of inflammation or membrane in the throat. The intestinal tract and other viscera were examined by the analyst, but no poison was found. Sections of brain, heart, lung, liver, spleen, kidney, small intestine and mesenteric lymph gland were examined microscopically. Examination of the heart revealed active myocarditis. There were degeneration and disappearance of muscle fibres and an intense interstitial cellular reaction comprising mostly lymphocytes and large mononuclear cells.

Though several types of infection may have caused this condition, it was most likely due to an unrecognized attack of diphtheria some weeks earlier. One assumes that while the myocarditis was developing the inflammation of the throat subsided.

Syphilitic Myocarditis: Laboratory Number 37591.—A half-caste aboriginal labourer, aged twenty-eight years, died suddenly. At the autopsy the government medical officer found, *inter alia*, a slightly yellowish patch about 1·2 centimetres in diameter at the apex of the heart involving the whole thickness of the left ventricular wall. To establish its nature he submitted it for histological examination. The muscle fibres here were largely replaced by fibrous tissue in which were accumulations of cells, mostly lymphocytes. Pronounced endarteritis and the location of the fibrosis around arterioles suggested that the condition was due to syphilitic inflammation rather than to coronary ischæmia.

Infarction.

In a certain number of cases of the type mentioned at the beginning of the paper, in which a coronary artery is narrowed but no acute changes are evident macroscopically, the use of the microscope may reveal early acute infarction and so verify the presumption of death from coronary disease. Or one may find infarction unexpectedly in a hypertensive heart.

Early Infarction of Heart: Post-Mortem Examination 1550.—Two and a half days after an operation for bilateral pterygia, and while still in hospital, a man, aged forty-six years, suddenly died. Autopsy revealed hypertrophy of the left ventricle, the heart weighing 510 grammes. The larger coronary arteries were soft-walled and patent. Some yellowish streaks could be seen in the myocardium. Microscopic examination revealed necrosis of muscle fibres and infiltration with polymorphonuclear leucocytes. Death was due, therefore, not to the overtaxing of a hypertensive heart, but to occlusion of a coronary artery not evident macroscopically.

Suspected Abnormality of the Conducting System.

A difficulty in the histological investigation of the heart is that the focus of disease which may have caused death by initiating ventricular fibrillation may be minute, and only a lucky selection of its site or the use of multiple serial sections would reveal it. When heart block or other abnormality of the conducting system of the heart is suspected, Shennan[5] recommends microscopic examination at three sites: (i) the sino-atrial node lying in the right atrial wall in the groove between the superior *vena cava* and the auricula; (ii) the cardiac septum between the mouth of the coronary sinus and the fibrous septum; this includes the atrio-ventricular node, the bundle of His and its two main branches; (iii) a portion of the ventricular wall close above the base of the anterior papillary muscle of the left ventricle; the Purkinje fibres will be found in the subendocardial tissue.

Unsuspected Coronary Disease.

Many cases could be quoted in which persons of middle age, apparently in perfect health, have died suddenly; in

which macroscopically the heart and other organs appeared normal; and in which, after full investigation, the only abnormal finding was, histologically, a little fine fibrosis of the myocardium or atheroma of a branch of a coronary artery. Such a case was the following:

Coronary Disease, Evident only Microscopically: Post-Mortem Examination 3268.—An air force officer (ground staff), aged thirty-five years, apparently in perfect health, was sitting after his evening meal in front of the radio. He was seen to roll his eyes and slump forward. He died within a few minutes. Autopsy revealed no serious disease. In the heart the main coronary arteries were soft-walled and wide and showed only flecks of yellow atheroma. There was no hypertrophy of the heart, which weighed 355 grammes, nor any other abnormality to be found in it. The lungs were very congested and œdematous, and a little blood-stained fluid was present in the bronchi. The other organs were normal apart from congestion. The stomach was distended with a large meal. The viscera were submitted to the analyst, but no poison was found.

Sections of cerebral cortex, medulla, heart (apex and anterior wall), lung, liver, spleen, kidney and ileum were examined microscopically. The only significant finding was in the heart. The myocardium was normal, but the intima of the descending branch of the left coronary artery showed much hypertrophy. It was, in the section, distributed evenly round the artery and did not appear degenerate.

The presence of well marked disease of this coronary artery suggested that a small branch might have become occluded and caused death from ventricular fibrillation. While this was only suggestive, it was consistent with the history, and the death was certified as due to coronary occlusion and acute heart failure. (One wonders if the distended stomach may have contributed in some way to the acute heart failure.)

Lungs, Liver and Kidneys.

Microscopic examination of lungs, liver and kidney in difficult cases occasionally provides significant information bearing on the cause of death. In the lungs may be found pneumonia, chronic congestion, or emphysema (which may have caused failure of the right side of the heart); in the liver, central congestion of lobules or some type of necrosis; in the kidney, vascular, inflammatory or degenerative changes, or the intercapillary glomerulosclerosis characteristic of diabetes. Often the final diagnosis is made, not from the appearance of one organ only, but by correlation of the evidence from them all, as well as from other sources. Some illustrative cases follow.

Coronary Atheroma, Fatty Liver: Post-Mortem Examination 3571.—A woman, aged thirty-six years, living apart from her husband and employed as a hotel waitress, was found dead in bed. She was frail and undernourished and had not felt well enough to work for the previous two weeks. For the last week she had complained of influenza, earache and pain in the left side of the chest. The autopsy did not demonstrate clearly the cause of death, but it showed the following pathological features. The brain was rather œdematous. In the heart, which weighed 300 grammes, the coronary arteries were wide, with small patches of atheroma. There was some chronic inflammation of the tonsils with pockets of pus. The liver was fatty and friable. The stomach contained some brown fluid smelling of beer. A suspension operation had at one time been performed on the uterus, and the appendix, left uterine tube and left ovary had been removed. The other organs showed no significant changes, nor was any injury or other abnormality visible externally.

The viscera were sent to the analyst, but he found no poison therein. The amount of alcohol in the blood was estimated and found to be 0·08%. This amount would not be significant as regards the immediate cause of death. The blood did not react to the Wassermann, Eagle or Kline tests.

Microscopic examination confirmed the fatty condition of the liver, suggesting chronic alcoholism. (Later one heard that she was, in fact, a "heavy drinker".) There was also some central congestion of the liver lobules, suggesting heart failure. Some fibrosis was found in the heart muscle and much atheroma in a branch of the coronary artery included in the section.

The cause of the death was ascribed to coronary occlusion. It was considered also that the vitality of her organs, especially the liver, had been damaged by chronic alcoholism.

Myocardial Failure; "Pus" in Bronchioles: Post-Mortem Examination 3307.—A man, aged fifty-five years, died after an illness of some hours' duration. The autopsy was begun twelve hours later. The heart was found to weigh 325 grammes. There was much atheroma of the coronary arteries, but their main trunks were nowhere completely occluded. The heart muscle seemed normal except for pallor in places.

Some creamy fluid resembling pus was present in the bronchioles of the lower lobe of the right lung, giving rise to a suspicion of bronchopneumonia. The lungs were also tough and dark red. The lobular centres of the liver were congested and sunken. The other organs appeared normal.

Microscopic examination clarified the cause of death as myocardial failure. It revealed small areas of fibrosis in the myocardium and some degeneration of muscle fibres. In confirmation of this, examination of the lungs showed chronic congestion, and that of the liver central congestion and necrosis of the lobules. There was no sign of pneumonia, and the "pus" in the bronchioles proved to consist of desquamated epithelium.

This case illustrates the observation that the creamy fluid often found *post mortem* in bronchioles (also in kidney pelves, bladder, uterus and uterine tubes) is a trap for the unwary pathologist. To the naked eye it closely resembles pus, but it should never be accepted as such unless the finding is confirmed microscopically. It often consists merely of an emulsion of epithelial cells, which in these sites appear to desquamate readily after death.

STEP 4. BACTERIOLOGICAL INVESTIGATIONS.

A mysterous cause of death is a fulminant infection that overwhelms the patient in the space of a few hours. When such an infection is suspected, an endeavour should be made to grow the responsible organism in culture. If the need is evident at the beginning of the autopsy, blood may be obtained aseptically from the heart with a syringe after the outer surface has been seared. At a later stage the interior of the spleen provides the necessary culture material.

Meningococcal septicæmia is an example of an infection that may be rapidly fatal. The duration may be too brief for any abnormal appearance to develop in the meninges, but the disease usually declares itself by widespread hæmorrhages in the skin and often in the suprarenals. My attempts to grow the meningococcus in culture from these cases have been mostly unsuccessful, probably through being too casual. A more determined effort, with more attention to the vagaries of this temperamental organism, would no doubt have been rewarded.

Other infections that may be rapidly fatal, especially in infants, are diphtheria and enterocolitis, and culture of organisms from material from throat and bowel should be attempted in obscure cases, as well as any other cultures suggested by the morbid findings or the history.

Tetanus is a disease in which we are almost entirely dependent on the history for diagnosis. The wound where the bacilli entered may have been minute and may have entirely healed before death, or the bacilli may have entered through the intestinal tract. Moreover, in my experience the bacillus has not been easy to grow in attempted culture, even in obvious cases of tetanus. Nevertheless any suspected wound should be examined for *Clostridium tetani* by smear and culture.

Sometimes the organism responsible for a rapidly fatal illness is a virus—for example, in encephalitis or in the cerebral form of poliomyelitis. No facilities have been available in Brisbane for a systematic attempt to isolate viruses in these cases, but I hope that the new Institute of Medical Research will in due course be able to do so. In several cases in which a neurotropic virus was suspected, Dr. F. M. Burnet was good enough to examine portion of the brain stem sent by air to Melbourne, but without success.

Of the serological tests, the Wassermann is the most useful, and it should always be performed in difficult cases. It has occasionally solved the puzzle of the autopsy

and in other cases has confirmed the macroscopic and histological findings.

Unrecognized Syphilis: Post-Mortem Examination 2921.—A man, aged fifty-seven years, had suffered from multiple "rodent ulcers" of the face for six years. In his disappointment that they remained painful and unhealed after radiotherapy, he drowned himself. The autopsy revealed bilateral chronic orchitis, but no aortitis. Positive reactions to the Wassermann and Kline tests revealed the nature of the "rodent ulcers" and why they did not respond to radiotherapy.

Myelopathy, Myocarditis and Pneumonia: Post-Mortem Examination 2908.—A boy, aged seventeen years, had convulsive seizures at the age of five years followed by paralysis of all four limbs. He became a bed-ridden cripple and developed gross scoliosis. His final illness began with a cold; next day he had pains in the chest and vomited, and on the following day he died. At autopsy scattered nodules of bronchopneumonia were found; the heart muscle did not look fibrous, but was tough to cut. The spinal cord was not examined. A positive Wassermann reaction suggested the nature of the nervous disease. (The Kline test result was, however, negative.) Histological examination revealed widespread interstitial myocarditis, presumably syphilitic. Microscopic examination of the bronchopneumonic nodules was also interesting; it revealed masses of lipoid material filling many pulmonary alveoli; the condition was, in part at least, lipoid pneumonia. The pneumonia and the myocarditis probably both contributed to the death.

Agglutination tests find occasional use.

Scrub Typhus: Post-Mortem Examination 2814.—A Japanese soldier, recently captured in New Guinea, became ill with fever soon after his arrival in Brisbane. No malarial parasites were found in his blood and he did not respond to quinine therapy. He died ten days after the onset of the illness before a certain diagnosis had been made. Autopsy revealed toxic spoiling of the organs, bronchopneumonia, an enlarged, soft spleen and a chronic gastric ulcer oozing blood. The illness was demonstrated to be scrub typhus by agglutination of Proteus OXK by post-mortem blood serum to a titre of 1/640.

STEP 5. REVIEW OBSCURE CAUSES OF DEATH.

The final step in the puzzling post-mortem examination, after all necessary specimens have been saved for special examination, is to review mentally those causes of death which give little or no post-mortem signs. Some of them have already been mentioned.

A man may die from concussion without any post-mortem evidence of injury to the brain or skull, or any external wound, although my series does not include such a case. In two striking examples of death from concussion that I recall—a boxer knocked out in a championship contest and a wicketkeeper struck by a cricket ball—the brain substance seemed normal and the skull was uninjured, but there was some subarachnoid hæmorrhage as well as superficial abrasions and contusions to indicate that violence had occurred.

Reflex Nervous Action.

There is a group of cases in which death has been due to reflex nervous action—from a slight blow on the larynx or neck, from a punch on the solar plexus, or from some interference with the uro-genital apparatus, such as the passage of a catheter into the bladder or of a sound into the uterus. Diagnosis in these cases will depend on a history of the circumstances, or on the finding of the sound *in situ*, or of a set-up that indicated the intention. Some illustrations follow to show that death from reflex nervous action is a real thing. They were not "problem" cases, for the attendant circumstances made the diagnosis obvious.

Blow on the Solar Plexus: Post-Mortem Examination 129.—A professional boxer, aged nineteen years, was sparring. In the second round he received a "left" to the solar plexus. He immediately sagged at the knees and was held up for a while and then laid down. Ten minutes later he was pronounced dead. He was in good physical condition and had fought ten rounds ten weeks before. At the autopsy there were an abrasion on the right temple and many small ones on the chin, the left cheek and the upper lip. The brain, apart from congestion, seemed normal.

There was a little bruising of the omentum in the epigastric region, and a little blood-stained fluid lay around. There was no obvious injury to any abdominal viscus. The lungs were intensely congested; there were three large ecchymoses up to 3·8 centmetres in diameter and a few subpleural petechiæ.

Death from Injection into Uterus: Post-Mortem Examination 979.—A married woman, aged thirty-five years, living apart from her husband, was found dead in the (empty) bath. Between her legs was a Higginson's syringe and a basin containing a 14·75% solution of toilet soap. Autopsy showed that she was pregnant with a fœtus 39·5 centimetres long, corresponding to thirty weeks' gestation. The membranes were unruptured, but there was a cavity containing a little altered blood between the membranes and the posterior wall of the uterus. The wall here was softened and discoloured brownish black. The placenta was attached to the posterior wall just above this part. There was no laceration of uterus or vagina. No abnormality apart from congestion was found in the other organs. (It is presumed that no froth was found in the heart to indicate air embolism, although the point is not specifically stated in the notes.) The stomach contained a recent, undigested meal.

Sudden death evidently followed the injection of soapy water into the uterus.

Reflex cardiac inhibition was no doubt responsible also for the sudden death which overtook several women during the criminal induction of abortion; their histories were concealed. Cardiac inhibition was also suspected in the following perplexing case.

Possible Cardiac Inhibition from Abdominal pain: Post-Mortem Examination 717.—A woman, aged thirty-one years, had one child, aged twenty months. Five months before her death she had been treated for menorrhagia, which had steadily diminished, and for anæmia. April 1, 1938, was a hot day (maximum temperature 94·1° F.). At about 6 p.m. she told her husband that she felt "knocked up". She appeared to him to be ill, but not seriously so. After dinner, at which she ate little, she washed up while he put the baby to sleep. Later she complained that she had slipped, but not fallen, while going down the steps. A severe pain had come on in the abdomen, which she attributed to her menses, due that night or the next day. It was the same sort of pain that she had had on those occasions, but more severe than ever before. It did not last long. She wondered if in slipping she had hurt herself internally. About 8 p.m. she told her husband that she intended to douche herself (as was her premenstrual custom) and then go to bed. Fifteen or twenty minutes later he heard her scream, as if in agony, went into the bedroom and saw her in bed unconscious, dying. She was in her night attire. He could not say whether or not she had douched herself. No douching was in progress at the time of death.

Autopsy showed that she was pregnant. (It is doubtful if she was aware of this.) The embryo was about one centimetre long and was intact. There was no detachment of membranes, no bleeding, no sign of injury. The cervix seemed normal. The only noteworthy features of the other organs were a small, fleshy thymus, congestion and œdema of the lungs and congestion of liver and spleen. No special investigations were undertaken, except that the viscera were sent to the analyst, who found no poison therein. The degree of anæmia was not recorded.

The cause of death remains uncertain. On the facts as far as we have them, the most likely explanation is cardiac inhibition caused by a severe spasm of pain. The autopsy did not demonstrate the cause of the pain; it was not due to douching, for the earlier attack came on before that would have been done; the deceased likened it to dysmenorrhœa but more severe; it may have been associated with uterine contractions. Ureteral colic is a possibility, but unfortunately the autopsy notes are silent as to the condition of the ureters.

Taylor[6] quotes a case of death from emotional inhibition of the heart which occurred in 1893, and Sydney Smith[7] states (without giving references) that several instances are on record in which a severe fright has caused death and no lesion of the heart or other organ could be found. In the diagnosis of these cases important evidence was the failure to find, *post mortem*, significant disease of the organs. One will be cautious about accepting a diagnosis based largely on negative evidence, especially in the older literature. No such case has come into my experience. However, as death from reflex inhibition of the heart from afferent vagal stimulation is well

authenticated, sudden death from emotional inhibition is conceivable.

Professor A. J. Canny informs me that he has performed autopsies on a number of patients who were convalescing satisfactorily after a surgical operation, and who died suddenly a few hours after being informed that they were well enough to return home. There was no undue exertion to explain the death, and no pathological lesions were found. Death was provisionally ascribed to ventricular fibrillation precipitated by emotional reactions, which are a recognized cause of extrasystoles of varying frequency in some apparently healthy individuals. It is not suggested that the development of extrasystoles leads directly to incoordinate myocardial contraction; but it is conceivable that the unduly frequent genesis of extrasystoles may so impair cardiac output that the coronary bloodflow falls to a point by which myocardial anoxia provides the adequate background for fibrillation.

In this connexion one is reminded also of the psychic deaths reported among primitive peoples from such procedures as "pointing the bone".

Electrocution.

In death from electrocution there are no characteristic internal signs. In 15 of 21 cases that I have studied burns were present externally; in the other six cases the diagnosis had to be based on the attendant circumstances. The following case was difficult:

Electrocution or Sudden Cardiac Failure: Post-Mortem Examination 3010.—A foundry worker, aged forty-five years, had worked practically all day with a portable electric grinder. A workmate borrowed it for a few minutes. Deceased then picked it up, and had carried it about six feet when he suddenly fell to the ground apparently dead. He lay supine with the grinder on his neck. His mate received an electric shock when he touched the grinder. Autopsy revealed no burns on the skin. There was some old fibrous thickening of the cusps of the mitral valve, which did not appear to have caused either stenosis or regurgitation. There was moderate atheroma of the coronary arteries, which were wide, and hypertrophy of the left ventricle. The heart weighed 480 grammes. There was congestion of lungs, liver, spleen and kidneys and mild anthracosis of the lungs. Histological examination revealed no abnormality of the myocardium and no significant abnormality of liver, kidney or lung (except anthracosis).

The diagnosis here had to be made between sudden cardiac failure and electrocution. The evidence for electrocution was that death occurred while the subject was handling a "live" machine. On the other hand the heart was abnormal; there was also a history of rheumatic fever. I signed the death certificate, perhaps rather arbitrarily, giving the death as due to electrocution.

Spasm of the Larynx.

Death from spasm of the larynx would be difficult to recognize without a history. One curious example of this condition came before me:

Death from Laryngospasm: Post-Mortem Examination 1709.—A male baby, aged two years, developed laryngeal diphtheria, for which tracheotomy was needed. The inflammation rapidly subsided and on the fourth day the tube was removed. On the eighth day the child became cyanosed, owing to laryngeal obstruction, and the tube had to be replaced. He was not able to do without the tube for six weeks from the time of its insertion. He could manage without it for several days, and then laryngospasm would ensue. Through the laryngoscope the larynx appeared normal and there was a good airway. The tube was out for two weeks before the baby was discharged from hospital. He remained in good health, eating and sleeping well, for a further fourteen days. In the evening he had a good meal and was washed; while being dressed he began to cry, had difficulty in breathing, stiffened and died. This was ten weeks after the illness began.

Autopsy revealed no obstruction to respiration. The scar in the trachea was neatly healed; it did not involve the first two tracheal rings. The lungs were œdematous. The other organs were normal except that the left kidney and ureter were congenitally absent. The myocardium, liver and kidney were histologically normal. *Corynebacterium diphtheriæ* could not be cultivated from the throat.

Pneumothorax.

Pneumothorax is a possible cause of an obscure death. If its possibility is appreciated at the beginning of the autopsy, it is readily tested for by the opening of the pleural cavities through water placed in the sulci formed when the skin of the thorax is reflected laterally; contained air will bubble out. Or an outward rush of air may be noticed when a pleural cavity is opened. But if pneumothorax is not thought of until later in the autopsy, the diagnosis can only be suspected by the marked collapse of a lung or by the presence of some underlying disease —tuberculosis, abscess, trauma, emphysematous bullæ *et cetera*—or by dryness of the pleura, and the actual perforation through which the air entered the pleural cavity may be impossible to discover.

Anaphylaxis.

Death from acute anaphylaxis is rare; it has been reported following bee and wasp stings, injections of serum and of various antigens, and even after ingestion of minimal quantities of foods to which the subject was allergic.[8] Such a death may be inexplicable without a knowledge of the history, for the post-mortem changes are not characteristic. They are described by Moore[9] as follows:

> The blood is dark red and unclotted. The heart is dilated. The lungs are voluminous, and the alveoli are generally or focally distended. The mucous membrane of the respiratory tract is edematous. The veins of the splanchnic region are dilated. In the terminal bronchioles and alveoli there is a moderate to advanced edema. The only reliable method of objective proof of anaphylactic death is the use of serum for a Prausnitz-Küstner reaction.

Asthma.

Death may be due to an attack of asthma. I have performed autopsies in two such cases, as well as on a third subject who had suicided during an attack. My recognition of the nature of the deaths was assisted by hints from the history. In the absence of a history, the diagnosis should be suggested by the emphysema of the lungs and by the plugging of bronchioles with mucus, and confirmed by histological examination of bronchioles.

Other Puzzling Deaths.

Puzzling deaths may also occur from epilepsy and *delirium tremens*. There are certain poisons which provoke no post-mortem signs and also elude analysis.

Death in Young Children.

The determination of the cause of death in young children tends to be more difficult than in adults. Their nervous systems are more readily upset reflexly, and they may die from comparatively slight causes. They are liable to convulsive seizures and to fulminant infections. An abnormally mobile tongue may be "swallowed" with fatal result.[10]

Simpson[11] has pointed out that bronchitis or bronchiolitis is a common cause of death in infants, that it can develop rapidly, and that microscopic examination of the fluid in the air passages is important in its precise diagnosis.

I have never certified death as due to *status thymico-lymphaticus*. No doubt children vary considerably in vitality, but I have not accepted enlargement of the thymus as evidence of a special susceptibility to sudden death from trivial cause.

A Problem in Diagnosis.

The scheme here outlined for dealing with puzzling autopsies has gradually developed over the years in response to the stimuli of successive difficulties. It is likely that the failure to solve some of the earlier problems was due to incomplete investigation. But there have been more recent cases which, in spite of the free exploitation of the resources of the laboratory, have defied one's diagnostic efforts. It is comforting then to recall Sydney Smith's experience that in some cases the most minute

post-mortem examination fails to show any cause of death.[12] Such a case was the following.

Post-Mortem Examination 2982.—On November 7, 1944, a single woman, aged thirty years, employed for the previous month in a doctor's rooms, was attending a funeral service in an undertaker's chapel, when she suddenly fell forward from where she was sitting onto the floor. This occurred at about 2.30 p.m. and she was dead before the ambulance arrived a few minutes later.

The autopsy was begun at 5.30 p.m. Her height was noted as five feet three and a half inches, her build as slight and her state of nutrition as poor. Two fine, recent puncture marks were observed in the left arm near the shoulder and one, not so recent, near the right shoulder. There was no other sign of injury. The blood was still fluid. The brain, heart and respiratory tract were normal. The lungs were rather dry. The liver was pale brown. The uterus was normal except for congestion of the endometrium and some erosion of the cervix. One ovary contained a *corpus luteum* six millimetres across. The stomach contained a few ounces of semidigested food; examination of its mucosa showed many scattered bleeding points. Apart from congestion the other organs were normal.

No cause of death being evident, the usual viscera were sent to Mr. Meston, as well as the skin and subcutaneous tissue from both shoulder regions, as the puncture marks suggested the possibility of hypodermic injections. (Some time before, another doctor's attendant had died from overdosing herself with morphine.) No poison was detected, but in the stomach were found salicylic acid, phenacetin and caffeine from an A.P.C. powder.

Histological examination was undertaken of medulla, heart, liver and both kidneys. The only significant abnormality was found in the liver. The lobules tended to be small and irregular and there was overgrowth of bile ducts. In a section stained with *Scharlach R* almost all hepatic cells showed fine granules of fat. This indicated both old and recent damage to the liver, though it did not seem of sufficient degree to have caused death.

The history of this young woman was inquired into most minutely by detectives, and as a result they were satisfied that death was due to some natural cause. Very little was disclosed to assist in explaining the death, but she was said to be rather frail-looking. The doctor in whose rooms she had attended said that she had left his employment for family reasons on the previous evening. He had treated her for lumbago and fibrositis six months earlier, but recently she had appeared in perfect health. She was not undergoing any form of hypodermic therapy; she was not a drug addict; she had no access to his drugs.

Nor did investigation of her movements on the day of death assist. She had left her suburban home at 7.30 a.m. and spent the morning in the city, calling at various places. At 9 a.m. she had had her hair permanently waved by a process using ammonia. It is probable that she did not have a substantial lunch. She took lunch from home, but did not eat it, nor had she eaten some purchased cakes. (One recalls that only a few ounces of semidigested food were found in her stomach *post mortem*.) She arrived at the funeral parlour in time for the service at 2.15 p.m. and appeared quite well as she talked to her friends. The collapse came quite suddenly when the service was almost over at about 2.30 p.m., just after she had said "I am not going to see Roy". She referred to the body of the deceased, who had suicided after a domestic quarrel. Roy and his wife had been close friends of hers.

Investigation of the chapel was resultless. It was fairly well ventilated. There was no escape of coal gas, or of sulphur dioxide from the refrigerator, nor was dry ice in use. No one else at the service was affected.

The day of death was warm; the temperature reached a maximum of 84·2° F., and at that time the relative humidity was 47%.

In reviewing this case, one observes first that the investigations should have been more complete. The urine was not examined chemically, the blood was not submitted for the Wassermann test and too few tissues were examined histologically.

The dramatic suddenness of the death suggests a cardiac origin. One can postulate ventricular fibrillation from some minute focus of irritation, but she was rather young for coronary disease and at least the main coronary arteries were free from it. The mechanism tentatively advanced above by Professor Canny to explain deaths following emotional reactions may be applicable here. Alternatively it is conceivable, though unlikely, that her sorrow caused a psychological inhibition of the heart.

Boyd[13] refers to hypoglycæmia as the only recognizable factor in certain cases of unexplained and unexpected death. It is, for example, a likely cause when alcoholics with huge fatty livers die suddenly, for in their hepatic cells there is no room for glycogen storage. In the present case one recalls that the subject was poorly nourished; her liver was fatty and probably not functioning well, although there was no history of alcoholism; she probably had little or no lunch on the day of her death; and that day was warm. All these would be consistent with a disturbed sugar metabolism and render hypoglycæmia a conceivable cause of death. The blood sugar content was not estimated.

I felt unable to certify the cause of death.

CONCLUSIONS.

When a post-mortem examination has been made and the cause of death has not been determined, the following procedures are recommended.

1. Make sure that the anatomical dissection has been complete and that no significant organ has been overlooked. In particular a close study should be made of the throat and neck.

2. Examine the urine for sugar and acetone bodies and the liver for iron. Submit the viscera for analysis for poisons and, if necessary, the blood and urine for the estimation of their alcohol content.

3. Select specimens for histological examination from cerebral cortex, medulla, heart, both lungs, liver, kidney, and any other tissue of doubtful morbidity.

4. If indicated, attempt cultures on suitable media from blood, spleen, throat, bowel, wounds; or inoculate laboratory animals. Preserve a sample of serum for the Wassermann test.

5. Consider, in relation to the case, those causes of death which give little or no post-mortem signs. These will include ventricular fibrillation, reflex inhibition of the heart, concussion, epilepsy, convulsions, laryngospasm, electrocution, tetanus, acute anaphylaxis, fulminant infections, diabetic coma, hypoglycæmia, certain poisons, and others.

There should be readily available in the mortuary the containers, culture media, instruments *et cetera*, that are necessary for the collection of material for any of the above-mentioned investigations that are indicated. And I do not consider a post-mortem room complete without a copy of Shennan's "Post Mortems and Morbid Anatomy".

ACKNOWLEDGEMENTS.

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REFERENCES.

(1) A. S. Taylor: "Principles and Practice of Medical Jurisprudence", Ninth Edition, 1934, Volume I, page 619.
(2) E. H. Derrick: "Acute Porphyria, with Reports of Two Fatal Cases", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1946, page 241.
(3) W. D. McNally: "Toxicology", 1937, page 661.
(4) J. L. Hamilton-Paterson and E. W. M. Johnson: "Post-Mortem Glycolysis", *The Journal of Pathology and Bacteriology*, Volume L, 1940, page 473.
(5) T. Shennan: "Post Mortems and Morbid Anatomy", Third Edition, 1935, page 105.
(6) A. S. Taylor: "Principles and Practice of Medical Jurisprudence", Ninth Edition, 1934, Volume I, page 328.
(7) Sydney Smith: "Forensic Medicine", Eighth Edition, 1943, page 45.
(8) E. Urbach: "Allergy", 1943, pages 236, 557.
(9) R. A. Moore: "A Textbook of Pathology", 1944, page 641.
(10) A. G. Watkins, writing in D. Paterson and A. Moncrieff's: "Diseases of Children", Fourth Edition, 1947, Volume I, page 433.
(11) K. Simpson: "Pathology of Sudden Death", *The Lancet*, November 22, 1947, page 745.
(12) Sydney Smith: "Forensic Medicine", Eighth Edition, 1943, page 43.
(13) W. Boyd: "The Pathology of Internal Diseases", Fourth Edition, 1944, page 333.

THE USE OF TRACHEOTOMY IN PHARYNGEAL AND RESPIRATORY PARALYSIS FOLLOWING DIPHTHERIA AND BULBAR POLIOMYELITIS.

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PARALYSIS of the pharyngeal muscles may occur as a late complication of diphtheria or as an early manifestation of bulbar poliomyelitis. When there is involvement also of the intercostal muscles or the diaphragm or both, a clinical picture is presented, the main features of which are as follows: (i) difficulty in swallowing, often amounting to complete inability to swallow; (ii) the collection of a pool of mucus in the pharynx and about the entrance of the glottis causing respiratory obstruction; this mucus may become thick and purulent and difficult to remove; (iii) inhalation of this mucopurulent secretion with increasing signs of obstructed breathing; (iv) weakness or paralysis of the intercostal muscles, or of the diaphragm, or of both; (v) a weak, ineffective cough; (vi) adductor spasm of the larynx, which may be caused by abductor paralysis, or by the irritant effect of the mucopurulent secretion in the larynx, which acts as a foreign body. In addition, in bulbar poliomyelitis there may be signs of central respiratory or cardio-vascular failure and of cranial nerve involvement.

Treatment.

The purpose of this paper is to illustrate the use of tracheotomy when the above-mentioned signs are present, and few details of general treatment will be attempted.

Wilson[1] in his original article in 1932 on respirator treatment in poliomyelitis suggested tracheotomy. Since then there have been many reports of the successful use of this procedure. The whole subject of bulbar poliomyelitis has been dealt with exhaustively in the report of the Poliomyelitis Research Commission of Minnesota.[2] In this report stress is laid on the importance of maintaining a clear airway. However, so far as is known, there are no previous reports of the application of this treatment to respiratory and pharyngeal paralysis following diphtheria.

The primary object is to maintain an unobstructed airway. Tracheotomy helps to achieve this in the following ways.

1. By by-passing the pool of mucus in the pharynx. Posturing of the patient and suction of the pharynx are temporary expedients which are partially successful. However, the necessary frequent repetition of the procedure gives the patient little rest and is often distressing.
2. By simplifying direct suction of the larynx and trachea.
3. By avoiding the use of the respirator in certain cases. The reasons for this will be pointed out later.
4. By making it possible for an efficient nurse to feed these patients by nasal catheter or a Rehfuss tube with less risk or distress.

Indications for Tracheotomy.

Many of the symptoms of these patients may be ascribed to simple anoxia; but the onset of grave signs can often be dramatically sudden. The picture of the patient almost drowning in his own respiratory tract secretions is not uncommon in poliomyelitis. On the other hand the mechanism of death is often obscure. Undoubtedly central cardio-vascular or respiratory failure is responsible for some deaths, and possibly a period of anoxia may hasten this central failure, or in other words, early relief of anoxia may delay the onset of central failure. The Minnesota report gives the following indications for tracheotomy:

> Recent onset of bulbar symptoms with evidence of progression of the illness; progressive difficulty in swallowing, or the accumulation of secretions in the throat; and mental changes, especially anxiety apprehension hyperactivity, confusion or euphoria.

These signs are regarded as indications for an elective tracheotomy:

> The appearance of laryngeal stridor, dyspnœa despite adequate chest expansion, cyanosis and severe encephalitic symptoms, indicates serious obstruction of the airway and requires emergency intubation or tracheotomy.

In diphtheria central failure is rare, but otherwise the same criteria for tracheotomy hold good.

The Use of the Respirator.

As a rule patients with pharyngeal paralysis do badly in a respirator unless tracheotomy is performed first. Aspiration pneumonia and atelectasis are fairly common. The respirator does not supply an airway. It may have the opposite effect, of increasing the obstruction by sucking mucus into the trachea.

To quote again from the Minnesota Research Commission's Report:

> A tracheotomy is essential in any of these patients who have to be put in a respirator, because the mechanical respiratory excursion will produce pulmonary œdema if there is any obstruction to the airway.

It will be shown, moreover, that the operation of tracheotomy not only relieves the obstruction, but in some cases renders respirator treatment unnecessary by giving the weakened respiratory muscle less work to do. In at least two of the cases which will be described it will be shown that the patients were able to carry on quite well, in one case with almost complete intercostal paralysis and in the other with almost complete diaphragmatic paralysis. If a respirator is needed, the tracheotomy tube can be kept clear of the respirator collar by means of a curved bar, which pushes the collar towards the suprasternal notch and gives ample room for manipulation of the tube.

Moistened oxygen can be given through the tracheotomy tube, and in the cases described no complicated apparatus was used. It has been suggested that a positive pressure respirator is needed to facilitate expiration, which might be embarrassed by the pressure of the oxygen. The writer has no personal experience of this.

The Operation of Tracheotomy.

It is not intended to give a detailed account of the technique of the operation of tracheotomy. The following general advice, however, may be helpful.

1. Do not attempt the operation while the patient is in the respirator.
2. Local anæsthesia should always be used unless the patient is *in extremis*.
3. A large incision is recommended.
4. During the operation keep the patient's neck hyperextended, with the chin pointing direct to the ceiling, and do not allow the head to deviate to one side or the other.

The post-operative management is most important. The essentials are considered to be the following: (i) the use of chemotherapy to reduce the risk of secondary infection; (ii) frequent suction of the trachea and bronchi by a rubber catheter; this may be done by the nurse; (iii) frequent instillation of a saturated solution of sodium bicarbonate into the tube, to prevent the formation of thick dried mucus in the trachea and bronchi; if this has already formed, its removal by suction will then be facilitated.

Reports of Cases.

To illustrate the above-mentioned points the following reports of five cases are presented.

CASE I.—A boy, aged two and a half years, was admitted to hospital on July 4, 1945, suffering from faucial diphtheria. In the sixth week of illness he developed signs of pharyngeal paralysis, with great difficulty in swallowing and some respiratory obstruction. Soon he developed signs of diaphragmatic weakness, and his colour became poor. Respirator treatment was tried with little benefit. Continuous suction of the pharynx was tried, but probably owing to anoxia, respirations became rapid and irregular

and it was impossible to synchronize his breathing with the respirator. Subsequently he was removed from the respirator and tracheotomy was performed with dramatic relief. His condition improved so much that further respirator treatment was not needed. Seven days after tracheotomy the power to swallow had partially returned and the tracheotomy tube was removed. He then made an uninterrupted recovery.

CASE II.—A girl, aged eight years, was admitted to hospital on May 13, 1947, suffering from severe faucial diphtheria. Early in the sixth week pharyngeal paralysis had developed, with complete inability to swallow. Indefinite signs of diaphragmatic weakness were present, and as her colour was poor, respirator treatment was tried. If anything, her condition was worse in the respirator, and after a few hours the treatment was discontinued. During the next few days she vomited all nasal feeds and was kept on an intravenous drip administration. Frequent suction of the pharynx and posturing gave temporary relief. Seven days after the onset of pharyngeal paralysis, examination with the fluorescent screen revealed complete immobility of the diaphragm. She became restless and apprehensive, with exaggerated and rapid intercostal breathing. The respirator was again tried, but again gave very little relief. Tracheotomy was then performed under local anæsthesia, and a quantity of thick mucus was removed from the trachea by suction. Respiration then became slower and easier, even though the diaphragm still remained immobile. Nine days after the tracheotomy she was able to swallow a little, and in ten days there was some return of diaphragmatic movement. Sixteen days after the tracheotomy the tube was removed, and she completely recovered.

The histories of these two patients with diphtheritic pharyngeal and respiratory paralysis serve to illustrate two facts: firstly, that patients with pharyngeal paralysis may be worse in a respirator, and secondly, that even when the diaphragm is completely paralysed, the intercostal muscles can carry on adequately so long as a clear airway is maintained.

CASE III.—A boy, aged eight years, was admitted to hospital on April 18, 1947, with symptoms of bulbar poliomyelitis present for two days. On his admission to hospital he was completely unable to swallow, neck and spine stiffness was present, and the cerebro-spinal fluid findings were typical of poliomyelitis. Twelve hours after his admission to hospital his condition had deteriorated, and in spite of posturing and of frequent suction of the pharynx, respiratory obstruction had increased. Adductor laryngeal spasm had also developed, as evidenced by laryngeal stridor and a change in the voice. He was extremely restless and his colour was poor. However, the respiratory muscles were working normally, and there was no indication for respirator treatment. Intubation was accomplished successfully, with relief of the adductor spasm. However, as the tube became blocked with mucus, it was decided to perform tracheotomy. This gave complete relief. The patient's colour and pulse improved and his breathing was comfortable. During the next few days respiration at times was shallow and irregular. This irregularity may have been associated with disturbance of central control. It is not possible to determine whether relief of the symptoms of anoxia prevented the further development of these signs. Oxygen was administered continuously through the tracheotomy tube, and the patient managed to survive this stormy period. Seven days after the tracheotomy he was able to swallow, and four days later the tube was removed. The palate weakness persisted for some time, but otherwise he made a complete recovery.

CASE IV.—A girl, aged five years, was admitted to hospital on August 28, 1947, suffering from bulbar poliomyelitis. She had severe palatal and pharyngeal paralysis, and the pharynx was filled with frothy mucopurulent material. However, her colour was good, and although she was apprehensive, she was not restless. At this stage an interesting feature was variability of the pulse rate. The usual limits were between 90 and 120 per minute, but occasionally there were bursts of rapid rhythm from 180 to 200 per minute, which lasted for ten to twenty beats. It would appear that there was some disturbance of central cardio-vascular control.

A few hours after her admission to hospital the intercostal muscles showed signs of weakening, and breathing became mainly diaphragmatic. Twenty hours after her admission to hospital restlessness and slight cyanosis became apparent, and tracheotomy was performed under local anæsthesia. Thick, greenish pus was evacuated from the trachea. Her condition improved, in spite of the fact that the intercostal muscles now appeared to be completely paralysed. She remained fairly comfortable for six days; but perhaps owing to the fact that penicillin therapy was discontinued a little early, the secretion in the trachea became copious and purulent. On examination of the patient under the fluoroscopic screen, it was found that the right side of the diaphragm was not moving, and it was therefore decided to use the respirator. In the respirator decided improvement occurred, although the trachea still became blocked frequently. Six days later the intercostal muscles and the diaphragm showed some return of power, and as the respirator collar had produced a pressure sore, she was taken out of the respirator. Twenty days after the tracheotomy the respiratory secretion had diminished considerably and the tube was removed. Her condition then improved steadily; but at the present time she still has pharyngeal weakness and is unable to swallow. There is also slight weakness in the shoulder, but otherwise she appears normal.

CASE V.—A boy, aged seven years, was admitted to hospital on September 20, 1947, suffering from bulbar poliomyelitis. He was completely unable to swallow, much mucus was present in the pharynx, he had some respiratory obstruction, and the diaphragm was almost completely immobile. Tracheotomy was performed shortly after his admission to hospital. Definite but temporary relief was obtained. Twenty hours after his admission to hospital intercostal weakness had developed and respirations were irregular. Respirator treatment was tried, but there was no improvement, and the patient died thirty-six hours after his admission to hospital. Death was considered to be due to central respiratory failure, which was progressive and unaffected by treatment.

Summary.

The value of tracheotomy is discussed in relation to the treatment of pharyngeal and respiratory paralysis associated with diphtheria and of bulbar poliomyelitis.

Two cases of pharyngeal and respiratory paralysis following diphtheria and three cases of bulbar poliomyelitis are presented. One patient died, and the recovery of the others is considered to have been influenced favourably by the operation of tracheotomy.

References.

(1) J. L. Wilson: "Acute Anterior Poliomyelitis: Treatment of Bulbar and High Spinal Types", *The New England Journal of Medicine*, Volume CCVI, 1932, page 887.
(2) Minnesota Poliomyelitis Research Commission: "Bulbar Form of Poliomyelitis", *The Journal of the American Medical Association*, Volume CXXXV, 1947, page 425.

AN OBSERVATION ON LICHEN URTICATUS AND LIGHT.

By J. M. O'DONNELL,
Perth.

IN Western Australia light sensitization dermatoses (apart from *lupus erythematosus*) are not uncommon. They usually take the form of itching papular and macular erythematous eruptions on the hands, arms, legs and feet, on the front of the chest and on the face. The dorsal surfaces of the extremities are more affected than the flexor surfaces as a rule. In a number of cases observed by me there has been a history of "infantile eczema" on the extremities. Inquiries from the parents of some of these patients elicited the information that the "infantile eczema" had been in the form of itching crusted spots, which at first looked like "hives". In some cases the eruption had disappeared when the primary dentition had finished, but in others the eruption persisted till the fourth year.

The description of the "infantile eczema" satisfied me that these patients, as children, had suffered from papular urticaria and not from true eczema. Having this in mind, I decided to experiment with some patients suffering from papular urticaria whom I had under my care. I tried the effect of protecting the exposed parts from the effects of light by the use of long sleeves and long "rompers"; I also administered appropriate doses of elixir of "Benadryl" and niacinamide. All patients improved, even if only to the extent of lessening of the secondary impetiginization.

In several cases the change was remarkable; new papular lesions almost ceased to appear; the itching lessened and the secondary infection rapidly came under control with antiseptics.

In one case I left one leg and one arm uncovered. The covered limbs rapidly improved, whereas the uncovered limbs did not. Even in the winter the patients were improved by this method; bandaging did not seem to have quite the same beneficial effect, possibly because in the case of infants it is often necessary to use *crêpe* bandages, which are hot.

All patients did not benefit to the same extent, but all were nevertheless improved, even without the niacin or "Benadryl". As the result of my observations, I am inclined to the view that light plays a part in producing the lesions in some cases of *lichen urticatus* (which, of course, is essentially an allergic disease). Possibly some patients who have had *lichen urticatus* as children may be more prone to develop light sensitization eruptions in later life.

PENICILLIN RESISTANCE OF STAPHYLOCOCCI

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WHEN penicillin therapy was introduced, it was soon realized that resistant strains of organisms normally sensitive to the drug were likely to be encountered. The danger that this acquisition of resistance to penicillin might become a property of infecting staphylococci has been stressed frequently.

In an examination of more than 100 strains of staphylococci isolated where penicillin was not in use, Spink, Hall and Ferris (1945) found that all were sensitive to penicillin. This was also found by North and Christie (1945) using a stock collection of 88 strains of coagulase-producing staphylococci collected before penicillin was used for clinical purposes. North, Christie and Rank (1946), in a study of 59 strains isolated from wounds during intensive penicillin treatment, found a considerable number of highly resistant strains. Barber (1947), in an examination of strains from 100 patients, found that 38 were penicillin-resistant; eight of these 38 were isolated from patients who had had no penicillin treatment.

In routine testing of staphylococci for penicillin resistance in this laboratory, it was noticed that a number of strains showed a resistance which was appreciably greater than that of normal strains. Many were from patients who had received no penicillin treatment, and in view of this it seemed likely that the penicillin sensitivity of the average infecting staphylococcus was already being affected by the general use of the drug. To examine this possibility strains were collected from patients who had no past history of penicillin treatment; staphylococci from all "doubtful" patients were excluded. As the use of penicillin both parenterally and in the form of lotions *et cetera* is so general, some difficulty was experienced in making a collection, which accounts for the small number (28) of strains used in this survey.

Of the 28 strains, seventeen were isolated from skin eruptions, three from urine, three from sputum, four from eye and ear infections and one from the throat. All were pigmented, coagulase-producing, hæmolytic and isolated at an early stage of the disease.

Sensitivity to penicillin was tested by the tube method. Ten millilitre quantities of nutrient broth containing increasing concentrations of penicillin were inoculated with a four millimetre loop from a twenty-four hour broth culture of the organism and incubated overnight at 37° C. The concentration of penicillin which just inhibited growth, judged by lack of turbidity, was taken as the end point. A known sensitive strain (F.D.A. 209) and a known resistant strain (Crotty) were used as controls.

The standard sensitive strain was inhibited in all tests by one-twentieth of a unit of penicillin per millimetre and the standard resistant strain by twenty units of penicillin per millilitre.

Of the test strains, sixteen were inhibited by one-twentieth of a unit per millilitre, one by one-tenth of a unit, two by one-fifth of a unit, three by one unit, three by ten units, one by eighty units and two by 160 units.

If one-tenth of a unit per millilitre is taken as the upper limit of sensitivity of normal strains, 11 of the 28 strains showed a resistance to penicillin which is definitely above this figure; six strains were able to grow in the presence of five units per millilitre—a concentration above that which can be achieved and maintained in the blood.

The number of strains used in this survey is relatively small, but the proportion of resistant strains is sufficiently high to be significant, especially when compared with the complete lack of resistant strains found in the early surveys.

Not all patients from whom resistant strains were isolated were treated with penicillin, and several failed to report after treatment, so that the records give no adequate picture of the success or failure of penicillin with resistant strains as compared with the sensitive strains.

In view of the results of Warmer and Amluxen (1945) and of North and Christie (1946), which showed that resistance *in vivo* paralleled resistance *in vitro*, the present tendency towards indiscriminate use of penicillin should be discouraged.

Summary.

An examination of 28 strain of *Staphylococcus aureus* isolated from patients with no history of penicillin treatment showed that eleven had a resistance to penicillin higher than that shown by normal strains.

Bibliography.

M. Barber: "Staphylococcal Infection due to Penicillin-resistant Strains", *British Medical Journal*, Volume II, 1947, page 863.

E. A. North and R. Christie: "Observations on the Sensitivity of Staphylococci to Penicillin", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1945, page 44; "Acquired Resistance of Staphylococci to the Action of Penicillin", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1946, page 176.

E. A. North, R. Christie and B. K. Rank: "Penicillin-Resistant Staphylococci in Wounds: A Report Based on a Study of 59 Cases of Infected War Wounds", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1946, page 43.

W. W. Spink, W. H. Hall and V. Ferris: "Clinical Significance of Staphylococci, with Natural or Acquired Resistance to the Sulphonamides and to Penicillin", *The Journal of the American Medical Association*, Volume CXXVIII, 1945, page 555.

H. Warmer and J. Amluxen: "Comparison of In Vitro and In Vivo Penicillin Resistance of a Strain of Hæmolytic Staphylococcus Aureus", *The Jornal of Laboratory and Clinical Medicine*, Volume XXX, 1945, page 419.

Reviews.

HISTORY OF ANÆSTHESIA.

ONE of the "Sigma" publications, which deal with science from the popular angle, Victor Robinson's "Victory Over Pain" provides a simple, readable and reasonably accurate account of the discovery and development of surgical anæsthesia.[1] Although it was written and produced in the United States of America on behalf of British sponsors, its style is on the whole commendably brief and clear, with few insults to English composition and virtually no obscurities in meaning. Unfortunately this high standard is not well sustained in the latter portion of the book.

The author carries his story in progressive fashion from ancient times to the present day. He makes reference to a much greater variety of classical and mediæval sources than has yet been employed in similar works on the subject. Many interesting quotations and illustrations accompany this discussion, which is admirably set out.

Commencing with Humphry Davy, a series of biographical essays on the various men who contributed to the final discovery is presented. The tendency to include circum-

[1] "Victory Over Pain: A History of Anesthesia", by Victor Robinson, M.D.; 1947. London: Sigma Books, Limited. Sydney: Walter Standish and Sons. 8¾" x 5¾", pp. 352, with illustrations. Price: 16s.

stantial speculations in some of these accounts does not detract from their general veracity. The claims of Long, Wells, Jackson and Morton are given impartial treatment, leading to the implied conclusion that the last of these was the effectual discoverer.

The adoption and progress of the discovery abroad are now traced by means of more short biographies, those dealing with Liston, Simpson and Snow being notable. In addition there are accounts of the mixed reception of the novelty in various continental countries. A lengthy and fanciful attempt to establish the priority of Guthrie, "the backwoods chemist", as the discoverer of chloroform is, however, not singularly impressive. And so, with some note of the alarming incidence of deaths under anæsthesia, especially with chloroform, the first 234 pages of the book are completed.

From this stage on the quality of the work deteriorates noticeably. One wonders if the untimely death of the author early in 1947 had something to do with this decline. Here begins the story of local anæsthesia, comprising but two biographies, one of B. W. Richardson, who, it seems, invented an ether spray, and the other of Carl Koller, deservedly famous for the introduction of cocaine into ophthalmology. The first of these employs the worst devices of popular journalism; an entirely irrelevant and undoubtedly garbled story of the awakening of Queen Victoria to learn of her accession to the throne of England is followed by some pathetic bedside admonitions of an ailing parent to her egregious son. These in turn give place to a questionable and tedious account of the first use of ether in Edinburgh, from which such trivialities as the date and the nature of the operation are omitted. Fortunately the section on Koller does considerable justice to others concerned in the development of local and regional analgesia.

Brief sections on various techniques now follow, including a rather condemnatory appraisal of twilight sleep. Endotracheal, rectal and intravenous anæsthesia are then usefully dealt with, while spinal and refrigeration anæsthesia are assessed in simple terms. The penultimate chapter deals with ethylene, cyclopropane and vinyl ether, perhaps with undue enthusiasm in the first and last cases. The final chapter, happily brief, belabours the horrors of curare with lurid inaccuracy. The book closes with an epilogue, dedicated to the Ether Monument in Boston. If somewhat turgid in its presentation, this does portray the drama and tragedy of anæsthesia in an arresting way. And now, with a short bibliography and a good index, the work is completed. Despite its limitations and defects, it is well worth reading.

AUSTRALIAN RHODES SCHOLARS.

AFTER the hiatus of war years we welcome the reappearance of "The Australian Rhodes Review".[1] It includes twelve articles dealing with varied subjects from U.N.E.S.C.O. to prisoners of war. Each gives the personal experience and opinions of some outstanding Australian. Educational aspects form the main interest, and the quality of the articles, as may be expected, is high. Of special interest is the article based on the report of the Oxford secretary and dealing with the accumulated experience of the performances at Oxford and after residence there of the Rhodes scholars themselves.

In the thirty-four years up to 1937 200 Rhodes scholars came from Australia out of a total of 2018. First or second class honours were attained by 84% (33% firsts)—a result equalled only by New Zealand—and this does not include a number of research degrees. This easily betters the performance of college scholars and exhibitioners from England. The States run fairly even, South Australia and Victoria rather "shading" the others. All round South Australia, in spite of its smaller population, has the best record. It also has sent the most medical scholars. With Florey and Cairns at their head these make no unworthy showing. Five medical Australians have attained professorships. We learn that of the total scholars appointed (American and Colonial) 30% have taken up education, 20% law and 7% medicine.

The long medical course does not so readily fit the conditions as set out by the trustees. One would like to see a more even distribution among the professions.

In the two wars 106 out of the 212 Australians selected up to 1941 saw active service and seven laid down their lives. Most of the others were debarred by age and almost all carried out some defence activity. The medical services claimed 28—an admirable effort.

Of the four ex-scholars who died in 1945-1946 we note the name of J. W. Horan, ophthalmic surgeon, of Perth.

The volume is well printed on good paper and is of special interest to those whose sons are interested in becoming Rhodes scholars and of the many who have Rhodes men as friends. It deserves general support and encouragement as one of the very few serious magazines of Australian interest written by Australians for Australians.

[1] "The Australian Rhodes Review", Number 5; 1946. Published by the Association of Rhodes Scholars in Australia. 1946. Sydney, London: Angus and Robertson, Limited. 10¾" × 7", pp. 164. Price: 3s. 6d.

MODERN DRUGS IN GENERAL PRACTICE.

"MODERN DRUGS IN GENERAL PRACTICE", by Ethel Browning, was favourably reviewed in its first edition (1940).[1] The second edition (1947) is again a remarkably good and practical *résumé*, both of established drugs, "which the general practitioner has tried and found not wanting", and of others closer to the borderland of present-day therapeutics.

Penicillin, the sulphonamides, gold, cardiac drugs, sedatives and hypnotics, drugs acting on the autonomic system, diuretics and urinary antiseptics, local anæsthetics and thiouracil, are all described succinctly and with a touch of experience.

The possibility of the oral administration of penicillin, especially in infants, is overlooked. The sulphonamide chapter lags a little; William Withering published "The Foxglove and Some of its Medicinal Uses" in 1785 (not 1735); gold has no place in the therapy of asthma; obsolete drugs should be dismissed in a sentence; the proprietary hypnotics should be more critically evaluated. No mention is made of streptomycin, propylthiouracil or DDT.

However, this is a valuable little book, and practitioner and student will be amply repaid for reading it.

DELAYED UNION IN FRACTURES.

"THE CAUSATION AND TREATMENT OF DELAYED UNION IN FRACTURES OF THE LONG BONE", by Kenneth W. Starr, carries on the tradition of publishing the winning Jacksonian Prize essay (1944), and in its layout it follows the usual pattern of such essays, being liberally sprinkled with quotations and having a bibliography of 715 references.[2]

The material studied by the author comprised a thousand fractures seen in hospital, of which some 450 were from gunshot wounds. Representative histological sections are included from a number of biopsies taken.

The structure and chemistry of normal bone and the cycle of bone deposition and bone resorption are fully discussed and illustrated by excellent photographs of biopsy sections.

The section on the anatomy of fractures, the anatomical structures involved, and the process of repair following fractures will be of particular interest to surgeons because the extensive review of the literature is illustrated by clear and excellent coloured diagrams as well as by photographs of sections of biopsies. Some of these diagrams would be invaluable in the teaching of students.

In the section dealing with the healing of infected fractures, reference is made to experimental work in which an experimental fracture in an animal was irrigated daily with lactic acid which kept the pH of the fracture site at 4·0. This resulted in a copious liberation of amorphous calcium.

Under the operative technique section in the main part of the work which deals with the ætiology and treatment of delayed union, it is recommended that non-absorbable sutures be used and that avascular bone be protected where possible. The author states that "it is our practice to afford the most widespread contact between the vascular soft tissue and the avascular bones". He advocates the drilling of avascular bone with a one-eighth inch drill at distances of one-half to three-quarters of an inch. In borderline cases, in which replacement of lost bone rather than rigidity is desired, he advises the use of a bone graft from the ileum.

The book is beautifully produced and well illustrated, but many may find it a little difficult to read. Both the author and the publishers are to be congratulated on a book which will do credit to Australian surgery.

[1] "Modern Drugs in General Practice", by Ethel Browning, M.D., Ch.B.; Second Edition; 1947. London: Edward Arnold and Company. 8½" × 5½", pp. 232. Price: 12s. 6d.

[2] "The Causation and Treatment of Delayed Union in Fractures of the Long Bone", by Kenneth W. Starr, O.B.E., E.D., M.B., B.S. (Sydney), M.S. (Melbourne), F.R.C.S. (England), F.A.C.S., F.R.A.C.S.; 1947. London and Australia: Butterworth and Company (Publishers), Limited. 9½" × 6½", pp. 248, with many illustrations, some of them coloured. Price: 53s. 6d.

The Medical Journal of Australia

SATURDAY, JUNE 19, 1948.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

A BRITISH MEDICAL ASSOCIATION REPORT ON MEDICAL EDUCATION.

THE subject of medical education has been mentioned in these columns on several occasions during the last twelve months. On July 19, 1947, reference was made to a brochure published by the General Medical Council of Great Britain in which it criticized the Goodenough Report and made recommendations of its own. The brochure was published in April, 1947, and the Goodenough Report was prepared in 1944. The recommendations of the General Medical Council were published in full in the issue of this journal that contained our reference to the brochure. On November 15, 1947, the attention of readers of this journal was directed to a survey on medical education in the United States and Canada by the Council on Medical Education and Hospitals of the American Medical Association. On this occasion it was suggested that a survey on medical education in Australia might with advantage be carried out. On January 10, 1948, it was suggested that the Federal Council of the British Medical Association in Australia should do something to set going an inquiry into medical education from the Australian point of view. It will be remembered that when this suggestion was brought before the Federal Council it was not welcomed. Councillors were reminded that the Parent Body was preparing a report on medical education and consideration of the suggestion from this journal was deferred until such time as the Parent Body's report had been published. This report has appeared and is available for discussion; two articles on it will be found in the *British Medical Journal* for May 29, 1948, a copy of which has been received by air mail. The report itself is a document of 151 pages and is of most absorbing interest. It will, of course, be studied by those who have to do with medical teaching, but it cannot fail to appeal to all practitioners with real interest in their profession. The moving spirit of the committee responsible for the report has by all accounts been its chairman, Professor Henry Cohen, Professor of Medicine in the University of Liverpool. The other twenty members of the committee included the senior office-bearers of the British Medical Association, the deans of two faculties of medicine and one ex-dean, several consulting physicians and surgeons, four general practitioners and a representative of the British Medical Students' Association. It might be thought that a committee of this size would be unwieldy; this cannot have been the case if the style and general workmanship of the document are any criterion. The committee obtained opinions and help from many sources—a list of 78 is published as an appendix. The recommendations, generally speaking, reflect the statement by the committee that reform of the medical curriculum is not only a compelling and urgent need, but that the required changes are more radical than is generally conceded. Defects in the curriculum have been considered in relation to (*a*) the basic principles which should underlie the construction of a curriculum, and (*b*) established general educational practice.

General agreement will be expressed with the statement that a medical curriculum can only lay the foundations of a doctor's education, and that it should stimulate him to remain a student all his life. The student must be trained in the science and also in the art of medicine, and there is no doubt that "sound habits and methods of study are the foundation of continued self-education". Of fundamental importance in the working of any medical curriculum is the selection of medical students. A general cultural and educational background is essential, but the difficulty lies in the discovery of young men and women who have suitable qualities. A discussion on this subject will be found in THE MEDICAL JOURNAL OF AUSTRALIA for January 20, 1945, at page 65. The Committee realizes the difficulty to the full. It discusses three questions—intelligence, personal character and the interest of the prospective student in a medical career. It also refers to methods of testing described by D. H. Smyth, Sub-Dean of the Faculty of Medical Sciences at University College Hospital, London, in the *British Medical Journal* of September 14, 1946, and favours a continuance of experimentation with the tests described by him. Character and intelligence are indeed not gifts bestowed equally on individuals, and there is justification for the contention that the suitability of the student to continue his course should be reviewed during his course. "After the first and again after the second year of study there should be a ruthless weeding out of those found to be unsuitable." Discussing pre-medical education, the Committee expresses strong disapproval of "specific vocational trends" in pre-medical school curricula. It agrees with the Goodenough Committee that general science should be taught in the schools to intending medical students as a non-vocational subject. It disagrees, however, with the view that the passing of a school examination might be accepted as fitting the student for admission to the "medical curriculum proper". It also disagrees with the Goodenough Committee's suggestion that the student coming up from school with a "good elementary grounding" in general science will need to spend relatively little time on physics, chemistry and biology in his medical course, and that such special training as is necessary can be given as part of, or in association with, the teaching in anatomy and physiology. This, it thinks, is ill-advised and denotes a failure to recognize the fundamental importance of a

thorough training in the methodology of science which can best be inculcated in a special course in science at a university standard and in a university atmosphere. "The fundamental purpose of this course in the basal sciences is to teach scientific method and to inculcate habits of clear and logical thinking." The Committee holds that the three basic sciences, physics, chemistry and biology, should be taught concurrently throughout the first year, and that the courses should not be specially adapted to the vocational needs of medical students, but should be fundamentally the same as those courses taken by other science students. Much the same idea as this was put forward in the British Medical Association's report on medical education published in 1934. Here it was stated that there are not two brands of physics, chemistry and biology, one for medical students and one for other students. The Committee would have the first year devoted to a study of the basic sciences; it regards as "unsound pedagogically and impracticable to operate" the Goodenough Committee's recommendation that the basic sciences should be taught in association with anatomy and physiology. If science becomes a part of general education, the Committee thinks that it should be possible to lift the level of the first year course to an altogether higher plane than it occupies at present. This part of the student's training is so important that it should be conducted by none but the most capable and discerning teachers. Some idea of the aim of the Committee may be gathered from the suggestion that the student will learn to seek habitually for the underlying physical phenomena which constitute the basis of biology.

This brings us to the "pre-clinical period" when the student studies anatomy and physiology. The Committee's views on this subject are clearly and vigorously expressed and cover a great deal of ground. The aims of the pre-clinical period are expressed by the Committee in the following terms:

(i) To provide for the understanding of the morphological, physiological and psychological principles which determine and influence the organization of the living body as a functioning unit.

(ii) To relate and interpret the structural organization and normal physiology of the human body and thus to provide the data on which to anticipate the disturbances of functions which will probably result from interference with normal structure and structural relationships.

(iii) To enable the student to recognize the anatomical, physiological, and psychological bases of the clinical signs and symptoms of disorder due to injury, disease, or maldevelopment.

(iv) Similarly, to help the student to understand the factors involved in the development of pathological processes and the possible complications which may arise therefrom.

(v) To give the student such knowledge of the pre-clinical subjects as will enable him ultimately to employ competently and rationally all ordinary methods of examination and treatment (including minor surgery) that may involve such knowledge.

If the teaching of the pre-clinical subjects is to be efficient, there must be constant correlation of structure with function. For example, a student handling a dried bone will receive an impression of rigidity and permanency; he should at the same time be taught that in the living body even the inorganic constituents of bone are continually being removed and replaced. "The instruction in anatomy should be planned to present to the student a sound knowledge of the structure of the human body based on the biological principles which form a background of such knowledge, due attention being given to the functional aspects." The Committee is opposed to the idea that the student should dissect only selected parts of the body. It regards dissection of the whole body as essential for the preparation of the student for his clinical studies, but thinks that the amount of topographical detail taught should be much reduced. Here the critic will wonder where sufficient bodies are to be obtained to allow of this extensive dissection—what is desirable is not always possible. The Committee does not think that physiology and biochemistry should be presented to the student as separate disciplines. They should be correlated to form a single unified course and this should be done by a committee of teachers of both departments. The reforms most urgently needed in the physiology course are two—first, the correlation with anatomy and the pre-clinical sciences, and secondly, a reorganization of the syllabus so that greater emphasis is laid on human physiology and less on animal experimentation. A probable reply to this by some persons would be that a great deal would depend on the wisdom of the teacher in charge of animal experimentation. The organic chemistry taught in the biochemistry course should, it is held, be only such as will help the student to understand the biochemical processes of health and disease and the general concepts which underlie them. The practical course in biochemistry should be designed to demonstrate on the qualitative side certain physico-chemical phenomena of biological import, and quantitative experiments should be confined to the least complicated techniques and those which illustrate principles. One important statement in this part of the report is that clinical teachers should emphasize the disturbances in structure and function which result in disease and that the staffs of clinical departments should include experts in pre-clinical subjects. The Committee thinks that the syllabus in psychology should be closely linked with the first year teaching of biology and with other pre-clinical subjects.

Our reference to the section of the Committee's report dealing with the pre-clinical part of the curriculum has been fairly full, mainly because it is that part of the curriculum which would-be reformers often attack with suggestions of simplification or omission in order that more time may be available for the clinical instruction of the student. In the Committee's report the clinical period is considered at great length and the most important part of the whole document is contained in two chapters headed "The Student and the Patient" and "The Integration of Medicine". It is to these two chapters that those who eventually secure copies of the report should pay most attention. The first statement is that it is a matter for regret and concern that the rapid advances in medical knowledge and the development of the resources of medical science during the last half-century have not resulted in a proportionate improvement in the general efficiency of medical practice. This statement is easy to make and difficult to refute. Perhaps we may be pardoned for wondering whether efficiency in general practice as we know it in Australia has lagged so far behind advances in scientific knowledge as the Committee's general indictment would indicate. But whether the statement in question is wholly true or not, the idea of the Committee

in regard to medical teaching is sound. This is that whatever the cost of reconstructing the curriculum may be, "we should return to first principles and so remodel the training of our students that they will base their future practice on an understanding of each patient as a 'whole', using the resources of the specialties as aids to diagnosis and treatment". To this end medicine must no longer be taught as though it was divided into separate compartments. Disease must be presented to the student as a disturbance or disequilibrium in the structure and function of the organism due to pathological agents of various types; the concept of diseases as clinical entities must be regarded simply as a "descriptive convenience". In the Committee's opinion the sole object of undergraduate teaching is not, as has so often been held, to train good general practitioners. Undergraduate study can be expected to do no more than give the student a foundation for his career, and this foundation is the same whether he is destined for general practice or the practice of a specialty. In the planning of a curriculum it is held that each aspect of medicine should receive its appropriate emphasis in the coordinated scheme. Each specialty should be treated according to its nature and each should be taught to the extent and to the standard appropriate to a basic training in medicine. It will not be possible to follow in detail the ways in which the Committee thinks that this may be done. Some subjects which are highly specialized, such as plastic and neurological surgery, will be dealt with as far as possible when the larger general subject such as general surgery is taught and then mainly in regard to the scope and potentialities of diagnosis and treatment. Other subjects such as social medicine and psychiatry "should be regarded as an integral part of all aspects of medicine and should as far as possible be dealt with in relation to these wider subjects rather than as distinct and separate courses". The Committee makes mention of the training of teachers to teach—a subject that has been discussed on more than one occasion in these pages.

It is to be hoped that this short review of the British Medical Association's report will stimulate interest in it. We do not expect to find great divergence of opinion on the general principles that have been stated by the Committee, but probably the suggested treatment of certain subjects will cause keen discussion, especially among those teachers whose specialties are in their opinion inadequately dealt with at present. One idea should be put forward. At the present time and with the present set-up many first rate practitioners are trained and teachers in many schools are doing sound and lasting work. The reason will be found in a statement by the Committee in its first chapter when it quotes one writer who held that: "The student and the teacher, not the curriculum, are the crucial elements in the educational programme." We all admit that progress in medical science makes changes in medical curricula necessary. If, then, it is possible to devise a logically planned curriculum that is based on the best ideas and ideals, the students and teachers who do well today, will do better in the future. To this end sight should not be lost of a suggestion in the last paragraph of the report that the establishment of an Association of Teachers in Medical Schools would be a fruitful venture.

Current Comment.

SNORING.

SNORING is an affliction for those who hear the snoring; it is also, as Ian G. Robin has pointed out,[1] often a distressing physical handicap. It may ruin a happy marriage, and in some parts of the United States it is considered justification for divorce. Robin has been able to find practically no literature on the subject. He mentions work by J. F. Strauss which was discussed in these columns on January 20, 1944. He has also gathered together some useful facts from personal observation and discussion with others similarly interested. He points out that noisy respiration during sleep may be produced during inspiration and expiration by various structures in the respiratory tract, but limits the term snoring to sounds made by vibrations in the soft palate and posterior faucial pillars during sleep. It is usually associated with mouth breathing, but sometimes with breathing through the nose. A short inspiratory snore through the nose with the mouth open may, Robin says, be called a "snort"; "some persons are addicted to a series of snorts". Snoring is involuntary; it stops as soon as consciousness is regained. According to J. Whillis the vibrating part in a snore is the thin edge or velum of the posterior faucial pillars. The important factors are the relative position of the soft palate and tongue and the texture of the velum, which depends on the tone of the musculature of the glosso-pharyngeal arch and on the thickness of the tissues, especially the mucosa. The position of the palate is regulated by the tone of the muscles and the amount of nasal airway. The position of the head seems to be important in governing the position of the tongue. The nervous factors which influence the tone of the glosso-pharyngeal musculature are still rather obscure. Apart from nasal obstruction, œdema of the palatal mucosa or loss of tone in the muscular velum may be due to slight pharyngitis, smoking, obesity, plethoric tendencies, allergic manifestations and possibly granulomatous conditions of the pharynx. From the practical aspect these causes of snoring fall into two groups—organic and functional (or "dysfunctional"), though some dysfunctional element is probably present in all cases; the first seldom occurs without the second. There are three main age-groups among snorers: the child, the adult and the elderly person. Most children are cured by removal of their adenoids and tonsils, the exceptions being mouth breathers whose condition is of "functional" origin and those with infective or allergic rhino-sinusitis. Adults usually start snoring because of some organic cause. Lack of tone makes the condition common in elderly people.

The treatment of snoring depends on preventing or managing a group of related causes rather than a single cause. Amputation of the uvula used to be a popular practice, but it gives little relief; control of nasal obstruction is important; surgical procedures will sometimes be necessary, but simple decongestive nasal drops instilled before retiring may be all that is required; "Benadryl" may help. Robin thinks that he and other surgeons may have helped many potential future snorers by their efforts at removal of tonsils and adenoids—the patients have been left with nothing but a fixed fibrous band instead of a "nice mobile soft palate and faucial pillars". Strauss achieved a result something like this by the injection of a sclerosing substance. Controlling the position of the head will prevent the tongue falling back; a cotton reel sewn into the back of the pyjamas effects this by preventing lying on the back. The position of the tongue, soft palate and/or jaws may be altered by means of breathing, swallowing and phonetic exercises, or by orthodontic splints. The mouth may be kept closed during sleep by special splints or by a piece of adhesive plaster across the corner of the mouth. Most of this treatment is only tentative and much remains to be learnt about this homely subject, but it is to be hoped that others will follow up Robin's lead.

[1] *Proceedings of the Royal Society of Medicine*, March, 1948.

Abstracts from Medical Literature.

PÆDIATRICS.

Cleft Palate and the Mechanism of Speech.

MICHAEL C. OLDFIELD (*The British Journal of Surgery*, October, 1947) describes the physiology of normal speech mechanism and discusses cleft palate speech defects. He makes a plea for the careful speech training of the cleft palate child after operative repair. In order to determine the exact position of the soft palate, lips and tongue during the articulation of speech sounds, he describes an ingenious method of radiography which involves coating the lips, tongue and palate with bismuth meal and injecting lipiodol along the nasal passages and in the upper and lower surfaces of the soft palate. Some descriptions of the methods used in speech training after repair of the cleft palate are also given.

Hæmophilus Influenzæ Meningitis.

ON the basis of twelve years' experience in the treatment of meningitis without intrathecal therapy, Archibald L. Hoyne and R. H. Brown (*The Journal of the American Medical Association*, February 28, 1948) claim that recovery is more prompt and complications are fewer if intrathecal treatment is omitted. They report a series of 28 cases of *Hæmophilus influenzæ* meningitis with 26 recoveries, in 21 of which no intrathecal therapy was used. All patients received sulphonamide, eighteen received streptomycin intramuscularly and seven streptomycin intrathecally; eleven were given type-specific serum by intravenous injection. The authors consider that serum is unnecessary if the dosage of sulphonamide and streptomycin is adequate. They state that they placed a high value on serum before streptomycin became available, but now believe that serum will seldom be used when streptomycin is available. Lumbar puncture should be used primarily for diagnosis. The average number of punctures performed on their patients was two.

The Heart in Normal Infants and Children.

NATHAN EPSTEIN (*The Journal of Pediatrics*, January, 1948) reports the results of clinical, fluoroscopic and electrocardiographic studies of 260 children who were under observation as part of a genetic study. Children with known or suspected heart disease were excluded. The children were examined clinically at least four times a year and fluoroscopic and electrocardiographic examinations were made once a year. The observations were continued for five years. During the first seven years of life over 50% and in the age span eight to fourteen years 66% of the patients had precordial systolic murmurs. In about half the children the murmur was limited to the second and third left intercostal spaces, and in the remainder was heard over the apex or the whole precordium. The murmur in most cases persisted throughout the years of observation, but varied in intensity and quality on various occasions. The limits of amplitude and duration of the *P* waves and *QRS* complexes and the amplitude of the *T* waves corresponded closely to accepted standards. However, 18% of these children presented tracings with some unusual sign or aberration, a finding that emphasizes the caution that must be used in the interpretation of electrocardiograms. One-half of the aberrations were *P* wave changes, the remainder being divided between *QRS* and *T* wave changes. The cardiac silhouette was examined in the posterior-anterior and oblique views. The shape was normal in the posterior-anterior view. In the left oblique view the angle of clearance of the left ventricle was approximately 50° in 90% of cases. In the remaining 10% sporadic angles of clearance of 55° were observed, especially in children with high diaphragms. In the right oblique view no evidence of enlargement of the left auricle was found in any child.

Emotional Factors in Convulsions of Children.

REYNOLD A. JENSEN (*The American Journal of Psychiatry*, August, 1947) studied in detail 22 patients, 12 girls and 10 boys, between the age of two and sixteen years suffering from convulsive disorders. Intrafamily tension, broken homes, parental conflict, sibling rivalry, and rejection or over-protectiveness by a parent were found to be frequent causes of anxiety In eleven cases the relationship between the onset of seizures and an experience heavily charged with emotion seemed apparent. Following treatment of the anxiety, 10 of the 22 patients remained free from seizures. Early traumatic experiences and difficulties at school or at home were discussed. The authors make a plea for the fuller use of psychotherapy in convulsive disorders, particularly in children. This form of treatment in no way militates against the use of anticonvulsant drugs, water restriction, or dietary measures.

The Course of Bronchiectasis in Children.

FINDLAY J. FORD (*Glasgow Medical Journal*, January, 1948) has reviewed the results obtained in the treatment of bronchiectasis in the Royal Hospital for Sick Children, Glasgow. The survey is based on the case records of a 60-bed unit in the hospital and covers the years 1930 to 1944 inclusive; it includes 94 cases, in 65 of which the diagnosis was confirmed by bronchogram. Permission for this investigation was not given in the remaining 29 cases and in these the diagnosis is regarded as suspected. In many cases there was a long history of cough when the patient first reported, and the author feels that a more thorough investigation of the underlying cause of chronic or recurrent cough would lead to the earlier and more frequent discovery of cases of bronchiectasis. From this survey it appears that age is an important factor in determining the outcome of the disease, for in young children the disease is sometimes rapidly fatal. Of children in whom the diagnosis was made before the age of six years, one-third died. This accounted for 88% of the total known deaths in the series. Treatment was surgical in suitable cases (patients with unilateral disease) and consisted of lobectomy or pneumonectomy. The operative mortality was 17%. In some children there was considerable post-operative morbidity due to failure to detect bilateral disease prior to operation. The author believes that surgery is the treatment of choice and should be advised unless the disease is hopelessly widespread. Of the 22 children on whom the operation was successfully performed, fourteen are well and free of cough. However, when operation cannot be undertaken there seems to be a reasonable expectation of life, unless the child is young. Indeed in 16% of the children in whom operation was not performed the pulmonary signs disappeared. Some others carried on a fairly active and unlimited life for years. Sixteen of the 26 children in the unoperated group are either apparently well or no worse after a lapse of an average period of seven years. Medical treatment, including the use of sulphonamide and inhalation of penicillin mists, is considered useful in treatment or prevention of acute exacerbations of infection in the lung. In addition to chemotherapy it included postural drainage, general hygiene, milk and cod liver oil. Of the 36 children not submitted to operation ten have died, contact with ten has been lost, ten are unchanged or worse and six are apparently well.

ORTHOPÆDIC SURGERY.

Chondromalacia Patellæ.

J. BRONITSKY (*The Journal of Bone and Joint Surgery*, October, 1947) describes *chondromalacia patellæ* as a circumscribed degeneration of the articular surface of the patella, evidenced by softening, fibrillation with eventual fissuring, and erosion of the cartilage. He points out that Freund in 1939 emphasized the belief that, under normal conditions, joint cartilage requires friction and pressure to remain healthy; any variation of these stimuli, either above or below the physiological optimum, if active over a long period, would be deleterious to the joint cartilage. The trend of present thought is that: (i) our knowledge of the physiology and nourishment of cartilage is still incomplete; (ii) degeneration of cartilage without trauma does exist; (iii) acute trauma or static deformities can influence the origin and progress of the disease; (iv) the question of constitutional disposition and heredity as ætiological factors is still unsettled. The earliest detectable lesions are most frequently located on the medial facet, less frequently in the centre, and occasionally on the lateral facet of the patella. The usual complaint is of pain, frequently accompanied by weakness and a tendency of the knee to buckle, especially during hill-climbing or walking up and down stairs. Repeated momentary locking of the knee may occur, but there is no true locking except in an advanced case when free bodies have broken loose from the surface of the patella. Frequently the knee aches. Crepitation of the patella is the most characteristic sign, but may be absent. Pain on pressure over the patella is often present. X-ray examination is almost of no help in making a diagnosis in the early stages. The author concludes that chondromalacia is a relatively frequent condition in both young and old. In all patients

who do not respond to conservative treatment surgery will produce definite and lasting relief from subjective symptoms. The surgery of choice is either partial chondrectomy or patellectomy. In the author's series good results followed in 75% of all cases in which chondrectomy was performed. The age of the patient has little influence on the choice of surgical treatment. Chondrectomy should be reserved only for those cases in which the patellar cartilage and surrounding tissue are mildly or moderately involved. If there is a severe involvement of the synovial tissue, excision of the patella will give best results.

Osteoid Osteoma.

I. PONSETI AND C. K. BARTA (*The Journal of Bone and Joint Surgery*, July, 1947) consider that, mainly because of the typical histo-pathological appearance of the lesion, the osteoid osteoma is a benign neoplastic formation, and that many cases, presented in the literature as proof that the osteoid osteoma is a lesion of inflammatory origin, are in reality examples of localized low-grade osteomyelitis and not of osteoid osteoma. The authors followed seven patients for over two years. One patient had osteoid osteoma in a rib, this being the first such lesion reported. A second had osteoid osteoma in the calcaneus. The lesions in the next two patients were in the proximal half of the tibia. The remaining three patients had involved the middle phalanx of the right index finger, the lower half of the left fibula, and the left ilium, just above the acetabulum. Three patients were males and four were females. Their ages ranged from fourteen months to twelve years. They gave histories of bone pain of slow onset, usually intermittent in character, not relieved by rest, and present from three months to one year. These symptoms followed minor traumata in only two cases. If the lesion was in the cortical portion of a bone and was located superficially, an expansion of the cortex of the bone appeared within two or three months after the onset of pain. On physical examination a small area of exquisite tenderness could always be detected, with, in five patients, slight local swelling. The patients had no fever, although a slight local elevation of temperature was noticed in two cases. Other effects were limitation of motion and muscle spasm, if the tumour was located close to a joint; limping when leg bones were involved; moderate regional muscle atrophy, if the condition was of long standing. The authors state that the osteoid osteoma appears on X-ray films as a small nidus-like, circumscribed area (0·5 to 2·5 centimetres in diameter), either uniformly translucent or mottled with irregular and sclerotic bone. It is almost always surrounded by reactive new-bone formation, which is of moderate character when the lesion is located in spongy bone, but which becomes very pronounced when the osteoid osteoma is located in cortical bone. Usually the older the lesion, the more marked becomes this reactive bone sclerosis. The authors believe that the histological picture of osteoid osteoma is unmistakable, although variations occur in the apparent aggressiveness of its cellular elements. The characteristic appearance is an irregular network of partly calcified osteoid trabeculæ, intermingled with vascular and very cellular connective tissue containing abundant osteoblasts and some osteoclasts. The osteoblasts and other mesenchymal elements may be of normal appearance, but are sometimes big, irregular and very numerous, with hyperchromatic nuclei which exhibit some mitosis. In the osteoid osteoma of the rib, the reactive periosteal bone was broken through in one area, although there was no invasion of the surrounding soft tissues. The osteoid osteoma is usually sharply demarcated from the surrounding sclerotic bone. Inflammatory cells are absent. No organisms have been grown from biopsy material. The authors conclude that this series indicated that the treatment of osteoid osteoma must be by an extensive block resection of the lesion, including a large portion of the surrounding sclerotic bone. The tumour and the symptoms have recurred in those patients whose lesions were treated by curettage or incomplete excision. The osteoid osteoma in the phalanx of one of the patients was shelled out twice, and recurred each time. On the other hand, in those patients whose lesions had been totally excised, the tumours did not recur and the symptoms completely disappeared.

Idiopathic Scoliosis.

G. E. THOMAS (*The Journal of Bone and Joint Surgery*, October, 1947) reports better results in fifteen cases of idiopathic scoliosis treated by recumbency, traction and lateral pressure on a Thomas spinal frame than those obtained from other methods. He states that resistance to correction is due mainly to structural changes in the vertebræ—changes indicated by the appearance in the X-ray picture of wedging of the vertebral bodies. On purely mechanical grounds this wedging may explain the resistance to correction of the lateral deviation, but it does not explain the greater resistance to correction of the rotation. Rotation, a complicated deformity, consists of two elements—the rotation of the vertebræ as a whole and the structural distortion of the various parts of each individual vertebra. The author points out that since the upright posture favours the progress of the deformity, recumbency is an important factor in its correction. The influence of gravity is eliminated by placing the patient on a Thomas spinal frame, fitted with a head piece. Although the usefulness of intermittent traction during the application of plaster jackets in suspension has long been recognized, the value of continuous traction in the treatment of scoliosis has been underestimated. Longitudinal traction is exerted on the spine by fixing the lower extremities by extension strapping to the foot pieces of the frame, and by applying weight traction to the head through a Sayre halter. To exert lateral pressure, special appliances have been devised by which the amount, direction and site of the thrust can be controlled. These are screw pressure pads attached to the horizontal bars of the Thomas frame The patient is put on the frame for two or three days so that he may become accustomed to it and then longitudinal traction is exerted. After two days the pressure pads are applied and are brought to bear gently on the trunk. The weights and pressure are increased gradually, the comfort of the patient being the governing factor. X-ray photographs are taken regularly. The nursing is simple. Release of the screws and weights once daily for skin toilet suffices. The author states that if the curve is too high in the thoracic region a pad cannot be applied opposite the point of maximum curvature and counterpressure is impracticable. Also a lumbar curve is not amenable to this form of traction because of the absence of any structures through which pressure can be applied effectively. The author observed that in every case the maximum correction obtainable was secured within eight weeks, and that no matter how much the forces were increased, or how long they were in operation after this period, no further improvement could be obtained.

Internal Contact Splint.

G. W. N. EGGERS (*The Journal of Bone and Joint Surgery*, January, 1948) states that the internal fixation of fractures has a definite but limited field of application in the treatment of fragment displacement. When an ordinary plate is used direct contact with the fragments is prevented, as clinical and microscopic observations of fracture ends of fragments of bone have shown variable absorption of the fracture surface. The plate then actually keeps fragment ends apart by a distance equivalent to the absorbed portion. The result may be delayed union or non-union and often the plate will bend or break. In an effort to eliminate this factor and to reproduce the contact of the fragment ends as observed in closed reduction, the slotted type of plate was designed. The author states that application of the slotted plate, which he prefers to call the internal contact splint to emphasize the fact that it is not applied firmly to the bone, is technically not so difficult as plate immobilization. The contact splint has many advantages. The fracture ends are easily placed together in exact approximation, the muscle pull maintaining the desired contact; placing the screws in the bone is therefore easier. Oblique fixation screws across the fractured ends are eliminated and the fracture ends do not receive additional surgical trauma. Impaction of the fragment ends is neither desirable, physiological nor necessary. Bone absorption of the fragment ends is automatically compensated for by longitudinal muscle tone; constant contact during the healing period is thus accomplished. Fewer screws are necessary for controlled fixation; therefore, less bone trauma occurs. The splint is flat, and pressure on the bone and periosteum is minimal. Contact is accomplished with the least mechanical difficulty by the permission of longitudinal motion in one or both ends of the splint. As the irregular fragment ends are held in contact by muscle tone, the stability of the fracture is secure, and torsion is controlled because the plate locks owing to physical forces. There is less stress on the splint to break or bend it at the fracture site, because the space between the fragment ends of the bone, due to absorption, is eliminated. Pressure in excess of eighty pounds has been applied repeatedly in the experimental laboratory to the long axes of bones, with no detrimental effect on the stability of the fracture site when the fragments are held in place by the splint.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the sixth session of the Australasian Medical Congress (British Medical Association) to be held at Perth from August 15 to 21, 1948, has forwarded the following information.

Return Railway Accommodation from Congress.

The Western Australian Government Railways office has guaranteed return rail accommodation to the eastern States. However, to provide this additional accommodation on the Western Australian and Commonwealth Railways it is essential that members of Congress who wish to return to their home States by train should immediately inform the Honorary General Secretary of Congress, 230, St George's Terrace, Perth, of the number of berths required and the dates.

Additional Baggage Allowance by Air.

To assist members of Congress wishing to bring dress clothes and academic robes, the Australian National Airways has kindly agreed to increase the baggage allowance to 45 pounds for each member. This is ten pounds greater than the normal allowance. This concession does not apply to members' wives, so that the total baggage allowance for a member and wife will be eighty pounds. Naturally, greater amounts of luggage may be carried, but the excess would be chargeable. All Australian National Airways offices have been advised. To receive this concession practitioners should inform the Australian National Airways that they are members of Congress when making their bookings.

Accommodation.

The Executive Committee believes that there is a number of practitioners who intend coming to Congress, but have so far given no notification of this intention. To assist in arranging satisfactory accommodation for all visitors, these practitioners are earnestly requested to inform the Honorary General Secretary, Congress Office, 230, St. George's Terrace, Perth, advising dates of arrival and departure, method of travel, and if being accompanied.

Medico-Legal.

TUBERCULOSIS AND WORKERS' COMPENSATION.

ON March 19, 1948, a judgement of importance to medical practitioners was delivered at Melbourne by the Workers' Compensation Board of Victoria. The case was one of Nurse "S" *versus* Epworth Hospital. The judgement was published in *The Australasian Insurance and Banking Record* of April, 1948, and is reproduced by permission of the editor of that journal. The judgement is as follows.

[4618/1947, March 19, 1948.]
(Judge Stretton, Chairman, Messieurs Parkes and Wilkinson, Members.)

Disease—Injury by Accident—Onset of Disease as Accident.

Applicant entered respondent's service as a nurse in 1944. She was then in good health. She lived in the hospital. Her work was of an exhausting nature. She became "run-down" in health. Her duties brought her frequently in close contact with tuberculosis sufferers. A person in a debilitated state of health is more vulnerable to infection by the tuberculosis bacillus than is a person in good health. In February, 1947, applicant became totally disabled by the onset of pulmonary tuberculosis.

HELD, that the state of disablement which applicant suffered was a personal injury by accident arising out of and in the course of her employment.

Stark (instructed by William S. Cook and McCallum) for Applicant.

Stafford, K.C. (with Winneke), instructed by Maddock, Lonie and Chisholm) for Respondent.

Reasons for Award.

In this case an award has been made in favour of the applicant. The reasons are these:

The formal question for the Board's decision was "whether the incapacity of the claimant, which commenced on the 10th day of February, 1947, was due to personal injury by accident arising out of or in the course of her employment". The evidence showed that applicant became incapacitated, and ceased work, on or about January 27, 1947. No point was made of the fact that the actual date differed from the alleged date.

The "accident" upon which applicant relied was alleged to have been a disabling onset of tuberculosis which she suffered and which incapacitated her. The real matter for decision was whether the onset was caused by her employment or otherwise; or, stating it more particularly in the words of counsel for respondent: "Did she catch it at the hospital or did she catch it elsewhere?"

Since 1934 the applicant had been working as a hospital nurse. She nursed in Melbourne until 1938. Then she began in London and continued there until 1943. She left England and joined respondent's nursing staff in 1944. During her stay in London she did not come into contact with known tuberculosis patients. From 1939 onwards each of the employees and nursing staff at the London hospital underwent at intervals of six months an X-ray examination of the chest, which was made for the purpose of detecting pulmonary disease. Her last examination was in May, 1943. No sign of tuberculosis or other pulmonary disease was revealed by any of the examinations. She reached Australia in January, 1944. She felt very well. Up to that time she had on the whole been free of colds.

She began as Senior Sister at Epworth in February, 1944. A little later she became Assistant Matron. She lived in the hospital except on her days off duty. The duties attaching to the office of Assistant Matron were, nominally, supervisory. In fact, because of an insufficiency of nurses, she spent at least two-thirds of her time in the wards with the patients, frequently assisting in tending them and necessarily coming in close contact with them. There were always acknowledged tuberculosis patients in the hospital. There are frequently in hospitals, generally speaking, patients who are tubercular, but whose tubercular condition escapes discovery. There appears to be no reason to suppose that Epworth did not have its share of such undiscovered tuberculosis sufferers. In addition there was, during the applicant's employment at Epworth, a nursing sister who later was stricken down by tuberculosis and who became a patient at the hospital. At times she became ill, whilst nursing; applicant was in close contact with her during her illnesses. After she was admitted as a patient, applicant was frequently in close attendance upon her.

The sister who was in charge of the ward in which the tubercular patients were kept resigned her employment late in 1945. She was not replaced, except intermittently, for short periods, by relieving sisters. Applicant, who normally visited the tuberculosis patients' ward every day, was, after this sister's departure, obliged to spend even more time, and to come in closer, intimate contact, with the tubercular patients.

Throughout her employment by respondent, applicant lived a very strenuous and exhausting life, the hardships of which were increased by the insufficiency of nurses, which added to her supervisory and office work the extra burden of active, physical assistance in the nursing of patients, which necessitated her being frequently in actual contact with them. Her health declined, she became subject to distressing colds and painful sensations in the chest, attacks of dizziness, sweating, loss of appetite and loss of weight. In January, 1947, she was obliged to cease work. She was admitted to the hospital as a patient. She was found to be suffering a condition of pulmonary tuberculosis. She has, ever since, been incapacitated by that condition, but is recovering and should soon be able to undertake intermittent spells of light work.

The evidence of Dr. J. E. Sewell, which was accepted by the Board, showed that the danger of contracting tuberculosis is a common one amongst hospital nurses, the danger emanating from the undiagnosed, unsuspected cases which occur in any hospital; that it is fairly common to be able to trace its cause to contact with a known case, but more common to be unable to find the source of infection; that the matters which determine whether one may become infected or escape infection are, chiefly, the subject's susceptibility to infection by the bacillus; the state of resistance of the subject to it; the "size of the dose" of the bacillus and whether the "dose" is repeated. Dr. Sewell emphasized the fact that a person who is in a "run-down" condition is more likely to become infected than one in good

health. He expressed the opinion that it was almost certain that applicant became ill of the disease while she was at Epworth. He explained that all people have the bacillus latent in them and are accordingly "infected", but suffer nothing by that infection; that it was probable that applicant had been so "infected" before she joined respondent's staff; that such a state of infection does not constitute illness; that the condition of infection which produces illness was not apparent in applicant until some time after she began to nurse at the hospital; and that she almost certainly suffered a disabling infection while she was at the hospital.

It is considered that the hospital managers would have been in a position to procure and call expert medical witnesses in opposition to the claim had they considered that the facts were such as to justify them in doing so. They called none. Their only evidence, given by the Matron of the hospital, did not contradict the evidence of the applicant which was all one way and of a convincing nature. The evidence showed that the applicant entered the service of the hospital when she was in good health; that she was frequently in close contact with tubercular patients; that the work was exhausting and likely to cause debilitation of her health; that after she had nursed under onerous conditions for some time her health began to decline until she became very "run-down"; and that she finally became totally disabled by the disease.

It is but a short and almost unavoidable step to the conclusion that she became infected, in the course of her employment, by contact with tubercular sufferers at the hospital, her vulnerability to the disease having been increased by her low state of resistance which resulted from her debilitated condition, which had been caused by the extraordinarily arduous service demanded of her by her employment.

The result is that the answer to the formal question is that the incapacity of the claimant was due to personal injury by accident arising both out of and in the course of her employment.

Post-Graduate Work.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

PROGRAMME FOR JULY, 1948.

THE Melbourne Permanent Post-Graduate Committee announces the following programme for July, 1948.

M.D. Part II, M.R.A.C.P.

A course in blood diseases will be conducted at the Royal Melbourne Hospital by Dr. R. P. McMeekin, assisted by Dr. John Bolton, on two afternoons each week from July 6 to 22.

A course in renal diseases will be conducted by Dr. Leslie Hurley at the Royal Melbourne Hospital on two afternoons each week, as follows: July 27, "Classification of Nephritis; Albuminuria"; July 29, "Acute Nephritis"; August 3, "Subacute Nephritis (Nephrosis-Type Nephritis)"; August 5, "Essential Hypertension"; August 10, "Chronic Nephritis"; August 12, "Uræmia".

Courses for Part I of Degrees and Diplomas.

Courses for Part I degrees and diplomas, which commenced in March, will be continued until August.

D.G.O., Part II.

A course for the D.G.O., Part II, will commence in July and be completed before the examination in early October. It will consist of lecture-demonstrations in gynæcology and obstetrics, pathology and bacteriology.

Demonstration at Geelong Hospital.

At Geelong Hospital, on July 14, at 8.30 p.m., Dr. John Horan will hold a demonstration on "Indigestion". Dr. M. W. Morris, "Belleville", Ryrie Street, Geelong, will make enrolments.

Demonstration at Camperdown.

At Camperdown, on July 31, at 8 p.m., Dr. A. J. M. Sinclair will conduct a demonstration on "Signposts in Nervous and Mental Disorders". Dr. G. Watson, "Malahide", Camperdown, will make enrolments.

Inquiries.

Inquiries should be made from the Secretary of the Melbourne Permanent Post-Graduate Committee, 426, Albert Street, East Melbourne.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Course for Diploma in Clinical Pathology.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a course suitable for candidates for the Diploma in Clinical Pathology will be conducted for six months beginning on July 12, 1948. Application to attend this course must be made before July 2.

Seminar in Medical Statistics.

Dr. H. O. Lancaster will conduct a seminar entitled "The Decline in Infant Mortality in New South Wales" on Wednesday, July 14, 1948, at the School of Public Health and Tropical Medicine. Dr. Grace Cuthbert and Mr. W. J. Willcocks will take part in the discussion, and those interested in attending are welcome.

Film Evening.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the following films will be shown at the film evening to be held at 8 p.m. on June 25, 1948, at the Stawell Memorial Hall, Royal Australasian College of Physicians, 145, Macquarie Street, Sydney: "Abdomino-Perineal Resection of the Rectum" (sound version), "Peritoneoscopy", "Atomic Physics" (documentary film).

All members of the medical profession are invited to attend, and further inquiries should be made from the Secretary of the Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney (telephone: B 4606, BW 7483).

Attention is drawn to the screening of the film "Atomic Physics", which is extremely interesting from the point of view of research.

Correspondence.

PNEUMONOKONIOSIS IN NEW SOUTH WALES COAL MINERS.

SIR: I am very grateful to Dr. D. Gordon for his letter published in the journal of May 22, 1948, in reply to my letter published on April 24, 1948. I am grateful for the public acknowledgement by a competent authority of the facts that:

(*a*) "X-ray findings in pneumonokoniosis do not necessarily have (in any individual patient) much bearing on the amount of disability present." (While I agree with Dr. Gordon that this fact has been accepted for quite a time and while still stressing that Rubin, a very competent authority, goes further as previously mentioned in stating that "X-ray appearances give no clue as to the disability of the worker", I trust that this acknowledgement will contribute towards a wide acceptance of this evaluation even beyond the borders of Queensland and into New South Wales.)

(*b*) ". . . that the law and possibly the public require more clear cut decisions than we can give them" (although I would prefer to substitute "need more clear cut decisions than we have given them").

The late Charles Badham's remarks in reference to the administration of "only rough justice" are, of course, well known to anyone conversant with the history of assessment of disability in pneumonokoniosis in New South Wales, but having had the good fortune to have had some official association with the late Charles Badham over a period of ten years and the privilege of many discussions with him on industrial medical questions, I am sure that he did not intend his remarks to be used as an excuse for maintaining the *status quo*, but rather as an urge to further research in an attempt to improve the available methods of assessment.

Dr. Gordon's remarks on the difficulties in differential diagnosis including the evaluation of "anxiety states" are common knowledge in their general application to all practitioners and in their special application to specialists in

their own speciality and are hardly germane to the present discussion.

In the last paragraph of his letter Dr. Gordon raises a very delicate question in his reference to the legal training of the chairman of the "Old" Pneumonokoniosis Board. However, it is a very pertinent question and must be dealt with in the interests of adequate discussion.

I would first make my position absolutely clear. I did not intend my letter published on April 24, 1948, to be in any way part of a controversy between the two boards, and must now make a very candid statement in that I personally consider the establishment of the "new" board has merely resulted in perpetuation of all the unsatisfactory features surrounding the findings of the "old" board and that the purpose behind the establishment of the "new" board might have more readily come to fruition by reorientation of the *modus operandi* of the "old" board. In fact rather than intending to enter into any controversy between the two boards, I am afraid I rushed into the breach created by the publication for the first time of the inner secrets of the *sanctum sanctorum*, but I no more wish to do myself the injustice of criticizing the acknowledged qualifications and attributes of any person involved in the discussion than Judge Stretton did when he stated on one occasion: "I feel we gave off sparks rather than discussed the matter, and I feel that thereby I did myself a grave injustice. It might seem more appropriate to say that I feel I have caused Mr. Phillips to suffer under some injustice, but that is not so, because ultimately, when one speaks in intemperate terms of the opinion of a gentleman of his undoubted intelligence, then it is the speaker who utters those terms who does himself an injustice."

With this clear understanding I think it can be fairly stated that, whatever the legal or medical qualifications of the members of the "old" board, it is apparent that there was little application of forensic principles to their deliberations when we see the persistent repetition in their findings of microscopic degrees of incapacity (down to one-third of 1%) and realize that these microscopic measurements were made with a yardstick.

I would finally like to correct Dr. Gordon's impressions from my original letter that I was suggesting the collection of "objective facts derived from measurement rather than a mass of clinical opinion". This interpretation is not strictly in accordance with my views—I used the word objective as opposed to subjective with the conception that in the "imponderable" cases an objective detail would, for example, be expressed as "in this case we are unable to make any accurate assessment of incapacity" or "in this case we are unable to decide whether the incapacity present is due to pneumonokoniosis or some other cause". Where, of course, actual well-founded methods of measurement are actually available, this state of "blank negation" (which the legal profession are quite capable of interpreting into some economic value with more equality and uniformity than "rough justice") would be obviated. This conception also regarded the method of using X-ray appearances in pneumonokoniosis as a measure of microscopic degrees of incapacity as a subjective phenomenon—not to be tolerated in a scientific procedure.

I still feel that the question of a proper forensic foundation to the deliberations of medical boards has not been disposed of. Let us consider for a moment what steps might possibly be enlarged upon in a forensic survey in an endeavour to convert the recognized "yardstick" of measurement into a more accurate instrument. From a close study of many board certificates I think it would be necessary to commence by suggesting that the first principle in any problem of administration is to "mind your own business". The "business" of a medical board, of course, is to assist in the administration of certain legislation.

It has been cynically said that the basic principle of legislation is expediency, but it is clearly accepted that the basic principles of administration are equality and uniformity of treatment. With attention directed to the persistent necessity for equality and uniformity of treatment, the board would, of course, be careful to express its findings in terms directly related to its current measuring rod so that any difficulties in administration of the legislation might be brought to the notice of the legislators to permit of necessary amendments. It would no doubt also impress the necessity for some indication in the certificate as to whether the opinions expressed were based upon fact, inference or conjecture, with the reminder that in resorting to clinical impressions "a guess cannot be based on a guess nor a guess on an inference, nor an inference on a guess, but an inference may be based on an inference, if the inference is a justifiable conclusion based upon competent evidence, either direct or circumstantial", and would be a measure to determine whether a guess had unwittingly entered into the formulation of an expressed opinion.

I have no doubt whatever that, with a recognition of only the two facts agreed to by Dr. Gordon, a forensic survey would have by now suggested some modification of the principle of a "suspensory award", probably based upon relative mortality-rating tables, for submission to the legislature, in an attempt to amend the legislation dealing with pneumonokoniosis, so that in administration it might be capable of more accurate interpretation with equality and uniformity.

Detail cannot be adequately discussed in the space available in correspondence columns, and for that reason I have tried to confine my remarks to principles. The suggestion I am making is that there is sufficient evidence to warrant a reorientation of the *modus operandi* of the Special Pneumonokoniosis Board and that this could best be achieved by attention to basic forensic principles.

The subjection of members to cross examination in open court would in my opinion involve a much longer evolutionary process than the inclusion of a legal member on the boards.

Yours, etc.,

"Craignish", I. A. D. GRAHAM.
185, Macquarie Street,
Sydney.

May 24, 1948.

SIR: In the original article on this subject by Dr. A. T. Nisbet, and in subsequent correspondence, a good deal has been written concerning the Pneumonokoniosis Medical Boards in New South Wales. As a member of the board which still examines applicants for compensation from the western and northern coalfields, I think it would be of interest to make known a few facts which became evident to me when comparing the system for examining individuals claiming to be affected by pulmonary dust disease in New South Wales with that in Britain, where I recently had several opportunities of observing the work of the Silicosis and Asbestosis Medical Board.

This board, which was established under the Silicosis and Asbestosis (Medical Arrangements) Scheme (1931), consists mainly of full-time medical officers of the Ministry of National Insurance, and is divided into six panels, one being located at each of the following centres: Cardiff, Swansea, Stoke-on-Trent, Manchester, Sheffield and Edinburgh. The members of the board work under the supervision of a chief medical officer, whose main function is to secure uniform standards of diagnosis and efficiency, and in cases in which there is doubt or disagreement amongst the members of the panel, the chief medical officer's opinion is final. The same board examines all eligible claimants for dust disease, irrespective of the industry of employment. This system contrasts noticeably with that in New South Wales, where, including one at Broken Hill, there are four different Pneumonokoniosis (including silicosis) Medical Boards, two of which are for the examination of coal miners. These four boards are constituted differently and work separately without coordination or uniformity, and whilst there is something to be said for a separate board at Broken Hill, there is no sound medical reason why the remaining three could not be replaced by a single authority to examine all the other cases, irrespective of the industry or district where they were employed.

Whereas in New South Wales the two medical boards which examine coal miners are required to state the medical condition found in the applicant and his fitness for work, the board in Britain only issues official certificates entitling the workman or his dependants to compensation benefits in the following circumstances: (*a*) when death, or (*b*) when total disablement has been caused by "the disease" (that is, silicosis or asbestosis or pneumonokoniosis, alone or accompanied by tuberculosis), and (*c*) when a workman, though not totally disabled, is suffering from "the disease" to such a degree as to make it dangerous for him to continue at his work. In these cases a certificate of suspension from further work is issued. Although there are no clearly defined standards or requirements as to when the disease is present "to a dangerous degree" (to use the more popular phrase), in actual practice the certificate of suspension is issued in cases of partial disablement.

Two other points which emerge from comparing the arrangements in both countries are that (i) the medical examination which coal miners in New South Wales receive is more comprehensive than in the case of their colleagues in Britain, where the board is not required to state on the certificate other conditions unrelated to the inhalation of

dust, and (ii) the standard of diagnosis is rather more generous in New South Wales.

I showed a group of approximately seventy X-ray films from New South Wales coal miners to most of the members of the Silicosis and Asbestosis Medical Board in Britain. Most of these films, according to New South Wales standards, showed nodulation due to pneumonokoniosis varying from a very early to an advanced degree. According to the standards used in Britain, where the term "reticulation" is freely used, rather than "nodulation", the latter being reserved for the radiographic appearances of so-called classical silicosis, several of our "very early" and even a few "early" cases would be regarded by a majority of the board members as showing no evidence of pneumonokoniosis. Further, judged on their X-ray films, some individuals who were certified as being partially disabled due to pneumonokoniosis in New South Wales would not be thus certified in Britain, the board there taking the view that even if the worker had some disability of apparently pulmonary origin the amount of dust disease seen radiographically would not be sufficient to account for the disability. The members claim that this more rigid standard is justified because the radiographic diagnosis is confirmed in many cases by the post-mortem findings of the lungs. Because of a provision in the Silicosis and Asbestosis (Medical Arrangements) Scheme requiring, except in certain cases, a post-mortem examination before the medical board may issue a certificate that death has been caused by pulmonary dust disease, either alone or accompanied by tuberculosis, the various panels of the board see a large amount of post-mortem material and the number of cases in which the lung pathology can be correlated with the X-ray appearances is relatively much greater in Britain than in this country.

Another interesting point of comparison is that in Britain, while the films are taken by private radiologists, the latter do not attend the meetings of the medical board, and little or no attention is given to their reports. Some members of the board take the view that the radiologist, if present, would prejudice the work of the board in making its examination and diagnosis.

With regard to the assessment of disability in pneumonokoniosis, the methods used by the board in Britain are not in any way superior to those adopted in New South Wales, and all members are agreed that the estimation of pulmonary disability when other pathological conditions such as cardiac or renal disease are present is extremely difficult. In this connexion the Pneumonokoniosis Research Unit of the Medical Research Council, in South Wales, is investigating, by means of experimental physiological tests, the problem of facilitating disability estimation.

Finally, in addition to examining claimants for compensation, the Silicosis and Asbestosis Medical Board conducts preemployment and periodical medical examinations of workers in certain industries, namely, the sandstone industry, the refractories industry and specified processes in the pottery and asbestos industries. If, on these examinations, the worker fails to comply with certain prescribed physical standards, or is found to be suffering from "the disease" or from tuberculosis, he is suspended from further work in the industry or process by the medical board, except under certain circumstances.

Yours, etc.,

GORDON C. SMITH.

Division of Industrial Hygiene,
New South Wales Department of Public Health,
93, Macquarie Street,
Sydney.
May 27, 1948.

SINUSITIS AND SHORT-WAVE THERAPY.

SIR: Dr. Wilkie Smith has raised many contentious issues in his recent article (THE MEDICAL JOURNAL OF AUSTRALIA, May 22, 1948). He is almost completely at variance with established fact and current opinion. It is hard to know where to begin in pointing out his inconsistencies.

His problem is sinusitis, yet his whole conception of the disease is vague. He makes no attempt to place his diagnosis on a sound basis, but decides that anyone with X-ray signs of sinus dulness and nasal symptom complex has sinusitis. The average patient with any nasal disorders comes along with the ready-made diagnosis of sinusitis. Such an attitude is reprehensible in the physician. I can find no positive evidence of sinus infection in any of his quoted cases. Proetz has shown that an allergic antral membrane may swell almost to the point of obliterating the lumen of the antrum and then return to normal in a few hours. There is no question of infection here, and more benefit will result from the institution of a proper allergic *régime*. Often there is infection superimposed on an allergic state. In order to diagnose infection one should see pus issuing from a sinus ostium, and find evidence in a sinus washing, or make a bacteriological examination of the pent-up secretion. One would expect that when resolution takes place following short-wave therapy pus should be seen streaming from the ostium of the affected cell. Dr. Wilkie Smith makes no such observation in support of his thesis.

Acute and subacute sinusitis almost invariably do well with vasoconstrictors or proof puncture. I do not see any need for a patient to be put to the expense of frequent short-wave treatment for several weeks.

Dr. Wilkie Smith states that sinusitis is commonly the primary cause of asthma. Nobody will deny that there is often a nasal factor, but it is only a small part of the asthma picture. In his first case report a short-wave treatment is credited with restoring the antral membrane to normal, yet a year later the patient had asthma. It is obvious that some cause other than the thickened antral membrane was at work.

Apparently Dr. Wilkie Smith is a "doctor who does not like the knife". He admits to receiving a good deal of inspiration in his attitude against surgery from the Hajek Clinic in Vienna. Hajek has been a foremost worker in the development of surgery of the nose and sinuses.

Most ear, nose and throat specialists these days consider themselves physicians as well as surgeons, and have a conservative attitude to surgery of the nose. Operations are restricted to removing gross obstructions, tissues the seat of irreversible pathology, and draining retained infected secretions. Would Dr. Wilkie Smith deny the soundness of these indications? The bad results of tonsillectomy are referred to. It is fair to state that these are due to poor operative technique, overlooked sinus infection, sino-nasal allergy, or to unnecessary operation. Dr. Wilkie Smith never does proof puncture. His view entirely ignores the accepted principle of surgical drainage of a suppurating cavity with retention of pus. Does he treat appendicitis, cholecystitis and brain abscess with short-wave therapy? I am sure he has no evidence to offer of infection arising from proof puncture. I have never seen it. Does an intractable infection always result if a dentist extracts a tooth and does he refrain from extractions for fear of it? Headache is said to be one of the most common symptoms of sinusitis. Most rhinologists will agree that chronic antritis is usually a painless disease. Headache occurring in nasal conditions is usually due to the associated nasal obstruction, or to sensitivity to food allergens. Dr. Wilkie Smith attributes his cures to the use of short-wave therapy, yet he also gives antibiotics and vaccines and excludes milk from the diet. In these circumstances it is not correct to say that the short-wave therapy has brought about the cure. Snoring in children is ascribed to blockage of sinus ostia. An elementary knowledge of anatomy of the nose will indicate that such a suggestion is fantastic.

With regard to vasoconstrictors, elaborate experiments have been carried out in America to prove that most of those commonly in use have no deleterious effect on human nasal cilia. I do not know how the cilia of the mussel or cod-fish behave. I am in agreement that prolonged use of nose drops is not good.

Dr. Wilkie Smith's article can be very misleading to those without specialized knowledge who seek help in the care of these admittedly difficult nasal disorders. Short-wave therapy has a place in the treatment of sinusitis, and is used by many rhinologists, but it fills a minor role. Personally I have been disappointed with it. Dr. Wilkie Smith does his machine an injustice by making such extravagant unsupported claims for it.

Yours, etc.,

R. J. WOOLCOCK.

141, Macquarie Street,
Sydney,
May 27, 1948.

SIR: Many will have cause to be grateful for the publication of Dr. Wilkie Smith's article on "Sinusitis and Short-Wave Therapy" in THE MEDICAL JOURNAL OF AUSTRALIA of May 22, 1948. His results compare favourably with all other methods.

I have obtained corresponding results in Brisbane adhering closely to the same procedure. Only in cases of obstruction due to extreme deformity have I found it necessary to resect the nasal septum.

During treatment many modifications are required in short-wave intensity, vaccine, and other treatment to deal

successfully with individual and seasonal variations and fresh virus infections. Treatment of sinusitis by this method is, therefore, no haphazard undertaking, but requires the constant supervision of an experienced clinician.

Yours, etc.,

149, Wickham Terrace, JOHN A. SHANASY.
Brisbane,
May 31, 1948.

SIR: In reference to Dr. Wilkie Smith's article published on May 22, while there is a lot to be commended in the ideas he expresses, I must protest at the representation of short-wave therapy as the complete answer to all types of sinusitis. The writer mentions surgical failures, but I can find no suggestion of failure of the short-wave and I have seen many of these; in fact, I have seen a number of chronic cases in which acute exacerbations followed short-wave therapy.

A letter to your journal cannot give full details of all my objections to the "one treatment" theory, but let us examine the exact therapeutic action of short-wave diathermy. It produces heat in the tissues at depth. Is anyone suggesting that this alone will cure all types of infection concerned in all sinusitis? In my opinion, and in the opinion of most of my specialist colleagues, short-wave diathermy has a very definite value, but is an adjunct, and only a moderate one, to other methods. I have a dim recollection of a panacea for chronic tonsillitis, namely, surgical diathermy, some years ago.

There is only one remark that I can make about sinusitis in general, and that is early diagnosis makes treatment easy, and when the diagnosis is early it is very seldom that short-wave therapy is required.

Yours, etc.,

"Craignish", R. H. BETTINGTON.
185, Macquarie Street,
Sydney.
June 1, 1948.

INDUSTRIAL MEDICAL SERVICES.

SIR: I am endeavouring to collect reliable and complete data as to the number of medical practitioners in Australia who give service in factories, business firms and other industrial establishments, in order to make a comparison between the medical facilities provided by private industry in this country and those in Britain and America.

I should therefore be greatly obliged if medical practitioners who are actively concerned in this type of work on either a full-time or part-time basis, or who are "on call" for emergencies only, would get in touch with me either by letter or telephone (telephone B 06 (Sydney), extension 977).

In each case I should particularly like to know: (i) The name of the factory *et cetera* where the medical officer gives service. (ii) The number of employees. (iii) The amount of time spent by the medical officer each week at the factory *et cetera*. (iv) Whether or not a trained nurse is employed by the management in the first-aid or medical section.

All information supplied in respect of individual factories *et cetera* will be treated as confidential.

Yours, etc.,

GORDON C. SMITH.
Department of Public Health,
New South Wales,
Division of Industrial Hygiene,
93, Macquarie Street,
Sydney.
May 31, 1948.

REPORT OF A SURVEY OF CHILDREN BORN IN 1941 WITH REFERENCE TO CONGENITAL ABNORMALITIES ARISING FROM MATERNAL RUBELLA.

SIR: The report of Dr. Patrick in THE MEDICAL JOURNAL OF AUSTRALIA concerning his investigations into congenital defects associated with maternal rubella contains several points of marked interest that I think are worth drawing attention to.

1. There was not one case of congenital cataract occurring among those subjected to such risk during the first sixteen weeks of pregnancy. This is in keeping with the occurrence of very few cases of congenital cataract attributable to rubella among 189 inmates of the Blind and Deaf School of Brisbane.

2. Of 129 children born of mothers who stated they had had rubella during pregnancy, 84 escaped damage altogether.

3. Most of the congenital abnormalities occurred amongst children whose mothers had been infected between the eighth and the sixteenth weeks of pregnancy, and yet 55% of these escaped too.

4. The almost complete absence of any congenital defects among the 1000 children of the same age whose mothers gave a definite negative history of maternal rubella.

In the School for the Blind and Deaf in Brisbane there are now 58 children born in each year, 1939 and 1941. In each group a history of maternal rubella could be obtained from only 37 mothers. The average normal yearly admission to the school is slightly under eight. This leaves thirteen children in each group over and above the normal yearly rate whose deafness cannot be attributed to maternal rubella.

Is there then another factor at work to cause these abnormal admissions for 1939 and 1941, or are they the victims of unrecognized rubella? And I would ask why have there not been similar occasional marked increases in the past records of this school, which go back nearly fifty years?

Yours, etc.,

538, Ipswich Road, L. P. WINTERBOTHAM.
Annerley,
Brisbane.
May 27, 1948.

DIAGNOSIS OF GASTRIC DISEASE: SHOULD RADIOLOGY OF THE STOMACH BE ABANDONED?

SIR: In your issue of May 15, 1948, you published an article by Dr. V. J. Kinsella entitled "Diagnosis of Gastric Disease: Should Radiology of the Stomach be Abandoned?". The title of this article is more than provocative, it is mischievous. The suggestion that radiology of the stomach should be abandoned is calculated to cause loss of faith among the general body of the profession and the public in what is undoubtedly the greatest single aid in the detection of gastric lesions. It will cause many people suffering from gastric symptoms to refrain from using a method of examination that is essential for diagnosis.

The radiologists do not claim that their methods cannot be improved, and variations in technique for their improvement are welcomed, but there are ways and means of giving such suggestions, and we strongly resent the method here employed.

Dr. Kinsella mentions Sir Hugh Devine as his teacher and inspiration. We should like to quote his own authority. *Vide* "The Surgery of the Alimentary Tract", by Sir Hugh Devine, Chapter XXVI, "Radio-Surgical Diagnosis", page 242. The opening and following sentences of this chapter read: "The culminating point in the diagnosis of a case of surgical dyspepsia is the X-ray examination. In most cases, where such examination is carried out by an expert radiologist, definite and reliable information is obtained."

On behalf of the Australian and New Zealand Association of Radiologists,

Yours, etc.,

JOHN O'SULLIVAN, M.D. (Melbourne), F.R.A.C.P., F.F.R. (England), D.M.R. and E. (Cambridge), President of the Australian and New Zealand Association of Radiologists.
Macquarie Street,
Sydney,
May 31, 1948.

STANDARDS AT THE UNIVERSITY OF SYDNEY MEDICAL SCHOOL.

SIR: The standards at the University of Sydney Medical School are more than a domestic matter of importance only to the people of New South Wales. Every Australian competent to judge is proud of this great medical school and its famous graduates. Until recently everybody has been confident that it would continue to set a good example to the other medical schools in this land. I am among those who feel this pride and felt this confidence. Perhaps,

therefore, although I am a graduate of the sister school in Melbourne, I may be forgiven for writing a second letter on this subject in order to emphasize two points.

The first is that my confidence in the future of the Sydney school has been shaken still more by reading Mr. Hugh R. G. Poate's courageous and temperate but nevertheless condemnatory comments in your issue of May 29. Nor have I been consoled by reading once again the optimistic impressions gained in some unspecified way by Mr. Howard Bullock, as described in your issue of May 8.

The second is that it seems reasonable to suggest that a profession which has publicly and properly condemned the *Pharmaceutical Benefits Act* on the score that it interferes with the efficient performance of their duties by the practitioners of today, must surely condemn with equal publicity and even greater force a standard of medical education which, as one of our honoured leaders, Mr. Poate, has shown, must decrease the efficiency of the practitioners of tomorrow.

Yours, etc.,

ALAN NEWTON.

272, Domain Road.
South Yarra,
Melbourne, S.E.1.
June 2, 1948.

THE PHARMACOLOGY OF JUNCTIONAL TRANSMISSION.

SIR: In reply to Dr. P. R. Bull I wish to state that, with regard to the use of the term "postganglionic cell", in actual fact Dr. Bull is correct, but I make use of this transferred epithet for the sake of brevity and distinction from the preganglionic elements in the cell. By these preganglionic elements I mean the terminations of the preganglionic fibres which are in juxtaposition with the ganglionic cell which gives rise to the postganglionic fibres.

Yours, etc.,

F. H. SHAW,
Associate Professor of Pharmacology.

Department of Physiology,
The University of Melbourne,
June 4, 1948.

Hospitals.

THE CENTENARY OF THE ROYAL MELBOURNE HOSPITAL.

A SPECIAL centenary number of *The Royal Melbourne Hospital Clinical Reports* will be published in June, 1948. It is greatly enlarged and contains articles of general interest by varied authors.

The list of contents is as follows: "The Management of the Paralysed Bladder", W. A. Hailes; "The Surgery of Common Anal Diseases", T. H. Ackland; "The Treatment of Varicose Veins", G. R. A. Syme; "Colitis", Geoffrey A. Penington; "The Problems of Penetrating Duodenal Ulcer", W. E. A. Hughes-Jones; "The Mechanism Involved in Anæmias due to Blood Destruction", John Bolton; Foreword to the film shown by Dr. J. Monahan Lewis entitled "The Clinical Manifestations and Treatment of Pyorrhœa Gingivæ"; "Trends in Anatomical Research", Professor Sydney Sunderland; "The Control of Gravity Shock by the Electrical Stimulation of Large Muscle Groups", Frank L. Apperly; "Allergy of the Respiratory Tract", Ivan Maxwell; "Indications for Aspiration Biopsy of the Liver", P. J. Parsons, H. W. Garlick and R. Motteram; "Congenital Heart Disease", C. H. Fitts and K. G. Grice; "Cardiac Pain", William Evans; "Malignant Tumours of the Œsophagus and Upper End of the Stomach", E. E. Dunlop and J. I. Hayward; "Hyperparathyroidism", E. R. Crisp; "Cerebral Lesions due to Ischæmia following Trauma to the Neck", E. Graeme Robertson; "Forty Years of Surgery at the Melbourne Hospital", W. G. D. Upjohn; "Twenty-Five Years of Pathology at the Melbourne Hospital", Professor MacCallum; "Lung Cysts (Excluding Parasitic Cysts)", John I. Hayward; "The Diagnosis and Treatment of Lumps in the Breast", Victor Hurley; "Industrial Injuries and Reparative Surgery", B. K. Rank; "The Value of Physical Medicine in Modern Treatment", L. T. Wedlick; "The Place of the Clinical History in Medicine", L. E. Hurley; "The Melbourne Medical Students, 1862-1944", Bryan Gandevia; "Solid Tumours of the Mesenteries", Sir Gordon Gordon-Taylor; "Research Activities in the Department of Surgery, the University of Sydney", Professor Harold R. Dew; "Mucins and Mucoids in Medicine", F. M. Burnet; "Some Aspects of Disease of the Thyroid Gland", Keith D. Fairley; "Man and His Hearing", John Shaw; "The Royal Melbourne Hospital and its Early Surgeons, 1841-1900", K. F. Russell; "The Development of Thyroid Surgery in Melbourne", Sir Alan Newton; "The Complications of the Common Head Injury", R. S. Hooper.

The cost of this volume is £1 1s., and as these will be limited publication orders for the magazine should be sent as soon as possible to Dr. L. G. Travers, 41, Spring Street, Melbourne, C.1.

The Royal Australasian College of Surgeons.

MEETING AT SYDNEY.

A SPECIAL MEETING of the Royal Australasian College of Surgeons will be held on June 25, 1948, in the Stawell Hall, 145, Macquarie Street, Sydney, at 8.30 p.m. The subject is "Surgical Complications of Pregnancy", and the speakers will be Professor B. T. Mayes (University of Sydney), Mr. J. W. S. Laidley (Sydney), and Mr. I. B. Jose (Adelaide). The discussion will be opened by Mr. P. L. Hipsley (Sydney). This meeting is open to all members of the medical profession.

Australian Medical Board Proceedings.

SOUTH AUSTRALIA.

DR. ERNEST ALEXANDER JOSKE appeared before the Medical Board of South Australia on January 13, 1948, to defend a charge of infamous conduct in a professional respect within the meaning of the *Medical Practitioners Act*, 1919-1946, Section 26(*d*), the particulars of which were as follows:

"You have committed all or some one or more of the following acts, viz.,

1. You issued prescriptions for morphine sulphate dated 1/9/46, 8/10/46, 17/10/46, 1/11/46, 11/11/46, 12/11/46, 28/11/46 and 30/11/46 respectively, and one undated, bearing the name of one Mr. Tucker (described in some of the said prescriptions as Mr. C. Tucker, Osbourne Avenue, Beulah Park), which were not intended for the use of Mr. Tucker, or required or received by him, he never at any material time having consulted your professionally but which were issued by you at the instance of, and given or sent by you to one Mr. P. G. Morton to enable the said Mr. P. G. Morton to obtain supplies of morphine sulphate for his wife, a person addicted to your knowledge to that drug, without the knowledge and consent of the Central Board of Health, thereby knowingly defeating the provisions of regulation 20A of the Dangerous Drugs Regulations published in the Government Gazette on the 29th January, 1942, at page 142.

2. You issued prescriptions for morphine sulphate dated 31/10/46, 9/11/46 and 22/11/46 respectively, and one undated, bearing the name of Mrs. Coleman (described as of Glenelg, or Broadway, Glenelg) which were not intended for the use of Mrs. Coleman or received by her, but which were issued by you at the instance of, and given or sent by you to one Mr. P. G. Morton, to enable the said Mr. P. G. Morton to obtain supplies of morphine sulphate for his wife, a person addicted to your knowledge to that drug, without the knowledge and consent of the Central Board of Health, thereby knowingly defeating the provisions of the abovementioned regulation.

3. You, being a person registered as a medical practitioner under the *Medical Practitioners Act*, 1919-1946, on divers occasions between the 30th day of August, 1946, and the 31st day of March, 1947 (both dates inclusive), prescribed a drug to which the *Dangerous Drugs Act*, 1934, and the abovementioned regulation applied, to wit, morphine sulphate, for the treatment of a drug addict, to wit, Mrs. P. G. Morton, whom you were treating in the course of your medical practice, without the written authority of the Central Board of

Health, knowing that you were required by the provisions of the said regulation to obtain the written authority of the Central Board of Health to do so."

At the conclusion of the hearing the Medical Board passed the following resolution: "Consideration was given to the evidence taken at this meeting, and after closely questioning Dr. E. A. Joske and hearing his answers and explanations the Board came to the conclusion that the charge against him of infamous conduct in a professional respect could not be sustained."

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

Pantle, George, M.B., B.S., 1947 (Univ. Sydney), Royal South Sydney Hospital, Zetland.

Perkins, Ronald George, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Broken Hill.

Pringle, Muriel Winifred Beryl, M.B., 1947 (Univ. Sydney), General Hospital, Brisbane, Queensland.

Rawle, Kenneth Thomas, M.B., B.S., 1947 (Univ. Sydney), Mater Misericordiæ Hospital, South Brisbane, Queensland.

Rofe, Owen Fulton, M.B., B.S., 1947 (Univ. Sydney), Royal Hobart Hospital, Hobart, Tasmania.

Rofe, Rosanne Lyle Fulton, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Bathurst.

Rogers, Norman James, M.B., B.S., 1947 (Univ. Sydney), 65, The Boulevarde, Strathfield.

Royle, Richard Arthur, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Goulburn.

Rugless, Margaret, M.B., B.S., 1947 (Univ. Sydney), 8, Seaview Street, Randwick.

Russell, John David, M.B., B.S., 1947 (Univ. Sydney), Willesden General Hospital, Willesden, London, England.

Sabiel, Kevin Norman, M.B., B.S., 1947 (Univ. Sydney), Base Hospital, Grafton.

Scott, Hilary James, M.B., B.S., 1947 (Univ. Sydney), 12, McLean Avenue, Chatswood.

Sheaves, Bruce Boyd, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Manly.

Sheils, John David, M.B., B.S., 1947 (Univ. Sydney), Base Hospital, Grafton.

Sheppard, Vincent Earle Moxey, M.B., B.S., 1947 (Univ. Sydney), Royal South Sydney Hospital, Zetland.

Spooner, Robert Dubois, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Manly.

Stephen, Henry Mitchell, M.B., B.S., 1947 (Univ. Sydney), District Hospital, Cessnock.

Sutcliffe, Muriel Mary Helme, M.B., B.S., 1947 (Univ. Sydney), General Hospital, Brisbane, Queensland.

Tillett, John Varnell, M.B., B.S., 1947 (Univ. Sydney), Royal South Sydney Hospital, Zetland.

Walters, David John, M.B., B.S., 1947 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Wilson, Jean Margaret, M.B., B.S., 1947 (Univ. Sydney), 15, Berry Street, Granville.

Wilson, Raymond Allan, M.B., 1947 (Univ. Sydney), Mater Misericordiæ Hospital, South Brisbane, Queensland.

Books Received.

"Restoration Exercises for Women: New and Revised Edition Embodying 'Stand Up and Slim Down'", by Ettie Rout (Mrs. Ettie Hornibrook), prefaces by Sir Arthur Keith, F.R.S., M.D., F.R.C.S., LL.D., and the late Dr. A. C. Haddon, M.A., Sc.D., F.R.S.; Ninth Edition; 1948. London: William Heinemann (Medical Books), Limited. 8¼" × 5¼", pp. 96, with many illustrations. Price: 7s. 6d.

"The Surgical Clinics of North America" (issued every two months); 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Chicago Number; 9" × 6", pp. 286, with many illustrations. Price: £5 10s. (cloth binding) and £4 12s. 6d. (paper binding) per year.

"Anatomy and Physiology, and Causes of Disease: For the Use of Students in Tropical and Subtropical Countries", by John P. Mitchell, C.B.E., M.D.; Second Edition; 1948. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 240, with many illustrations. Price: 5s.

"The Metabolic Brain Diseases and their Treatment in Military and Civilian Practice", by G. Tayleur Stockings, M.B., B.S., D.P.M.; 1947. London: Baillière, Tindall and Cox. 8¼" × 5¼", pp. 270. Price: 16s.

"The 1947 Year Book of Pediatrics", edited by Isaac A. Abt, D.Sc., M.D., with the collaboration of Arthur F. Abt, M.D.; 1947. Chicago: The Year Book Publishers Incorporated. 7" × 4¼", pp. 454, with illustrations. Price: $3.75.

"Psychodrama", by J. L. Moreno; First Volume; 1946. New York: Beacon House. 9" × 6", pp. 448, with illustrations. Price: $6.00.

"Group Psychotherapy: A Symposium", edited by J. L. Moreno, M.D.; 1945. New York: Beacon House. 9" × 6", pp. 306. Price: $6.00.

Diary for the Month.

JUNE 22.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 23.—South Australian Branch, B.M.A.: Annual Meeting.
JUNE 23.—Victorian Branch, B.M.A.: Council Meeting.
JUNE 24—New South Wales Branch, B.M.A.: Branch Meeting.
JUNE 25.—Queensland Branch, B.M.A.: Council Meeting.
JULY 1.—South Australian Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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